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PAPERS TO BE PRESENTED
BEFORE THE SECTION ON

OPHTHALMOLOGY

OF THE

AMERICAN MEDICAL ASSOCIATION

Atlantic City, June 8-11, 1909

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These papers, being the property of the American Medical Association, are not released for publication. They are here printed solely for the use of the members of the Section on Ophthalmology.

TO THE MEMBERS OF THE SECTION ON OPHTHALMOLOGY

OF THE

AMERICAN MEDICAL ASSOCIATION

All the completed papers for the program of the Section on Ophthalmology of the American Medical Association, Atlantic City Session, 1909, will be found herein, and, in most cases, in the order in which they will be presented for discussion. Provision has been made for notes on the margins of the pages and on the blank page following each paper.

Folowing the plan in force during the last few years, the papers will not be read before the Section but each author will be allowed not to exceed ten minutes in which to summarize the points in his paper and to introduce discussion. The member appointed to open the discussion of any paper will be allowed not to exceed ten minutes and subsequent speakers five minutes. The official stenographer requests that the manuscripts of all written discussion be turned in to him.

The officers and executive committe desire to thank the essayists for their cooperation in making it possible to issue the presession volume on time.

ALBERT E. BULSON, JR., Secretary.

ALVIN A. HUBBELL, Chairman.

THE BOUND TRANSACTIONS

All of the within papers, together with the formal discussion of the same at the Atlantic City session, will be printed and bound in cloth, thus constituting the Transactions of the Section on Ophthalmology of the American Medical Association for 1909. Members of the Section and others may obtain these bound Transactions at $1 a volume by sending subscriptions to the American Medical Association, 103 Dearborn Avenue, Chicago, on or before July 1, 1909. After the volume is published the price will be $1.50.

ALBERT E. BULSON, JR., Secretary.

Transactions of the Sections of the American Medical Association

Since only enough books are printed to supply the demand, many physicians, who have not attended the Association sessions, or who have neglected to send in their subscriptions before the books were published, have been disappointed in not being able to secure the Transactions desired.

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REPORT OF THE COMMITTEE ON COLLECTIVE INVESTIGATION CONCERNING THE OCULAR MUSCLES *

LUCIEN HOWE, M.D., BUFFALO; AND HOWARD F. HANSELL AND THEODORE B. SCHNEIDEMAN, OF PHILADELPHIA

Almost immediately after the chairman of this committee was asked to undertake the work he associated two others with himself, Dr. Howard F. Hansell of Philadelphia, and Dr. Edward Jackson of Denver. The latter found it impossible to continue on the committee, and later his place was taken by Dr. T. B. Schneideman of Philadelphia.

The first step was to ascertain who were at all interested in such a study. Accordingly a circular letter was issued to each member of the Section on Ophthalmology. A copy of this letter is here appended as Exhibit A.

The total number of replies received to this was one hundred and twenty-five. It was gratifying to find that every member thus replying expressed approval of the effort, although forty found it impossible for various reasons to join in the study. An examination of these replies and certain considerations led the committee to decide that it was advisable to study several unanswered questions concerning the anatomy and physiology of the muscles, before attempting any cooperative investigation relating to their pathology.

Accordingly, a series of twenty-four questions was drawn up with considerable care, and different groups of the members were asked to investigate these different points. The letter explaining more in detail the scope and the general outline of the work, is also attached with this report, and is marked Exhibit B. Several of the members did not find the questions which were assigned to them entirely of their taste, and were accordingly asked to take other questions for study.

In making these arrangements, the committee has therefore sent out about 850 of the first circular letter and 85 of the second circular letter, and in addition has written thus far about 130 letters.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

As a result, however, we have now over 80 members who are studying with more or less exactness these different questions concerning the anatomy and physiology. It is probable that a number finding that any real work demands time and patience will promptly give up the effort; but it is also probable that some will persevere, and we shall be able in this way to establish a certain few underlying facts. These, with other data which we already have, will give a broader and firmer foundation for any study of the pathology of the muscles, which may be elaborated by members of our section or by others in the future.

EXHIBIT A

AMERICAN MEDICAL ASSOCIATION

Section on Ophthalmology

COMMITTEE ON THE COLLECTIVE INVESTIGATION OF QUESTIONS CONCERNING THE OCULAR MUSCLES

183 DELAWARE AVE., BUFFALO, N. Y., December, 1908.

My Dear Doctor:—At the last session of the American Medical Association, Dr. Wilder, then the chairman of the Section on Ophthalmology, advocated the same system of collective investigation for that Section as had been adopted with advantage in other societies.

In acordance with that suggestion Dr. Alvin A. Hubbell, present chairman of the Section, has constituted the undersigned a committee for the collective investigation of questions concerning the ocular muscles. For the sake of uniformity this committee will not only select the question or questions for study, but arrange other details of the work. If sufficient interest is shown in the subject by members of the Section, various phases of different questions could be studied by different subcommittees. In any case, however, the results obtained by each individual would be kept separate. The object is not to deal with theories, but to collect FACTS, which are carefully observed and accurately recorded. As every ophthalmologist meets daily with unsolved problems concerning the muscles, perhaps some data can ultimately be obtained which would be useful to all, without great effort on the part of any one.

The committee hopes to keep the bibliography of the subject complete, and to make it useful to students whether they cooperate in this investigation or not.

The Section will probably confirm the action of the Chairman in making permanent these committees on special investigation. If that is not done, the work could continue just the same, and if the results are of any value, ample opportunity would be afforded for publication.

Instead of confining the effort to a few, it seems best to issue this circular letter inviting the cooperation of every one in the Section. Among the younger members, especially, there are probably many who have not only the time and energy, but the training necessary for such work.

If, therefore, you are sufficiently interested to cooperate, even to a slight extent, please answer the following questions, and mail them in the enclosed envelope before the middle of December.

LUCIEN HOWE, Buffalo N. Y.
HOWARD F. HANSELL, Philadelphia.
EDWARD JACKSON, Denver.

1. Do you care to take any part in the proposed collective investigation of some question concerning the ocular muscles?
2. What suggestion have you to make if any, as to which aspect of the subject could be studied first to best advantage?
3. Have you any preference as to exactly which part of that question or any other you would like to have assigned to you?
4. Have you opportunities for work if desired, in some well-equipped laboratory? If so, is that physiologic, pathologic, ophthalmologic or of what kind?
5. Are you connected with any hospital or dispensary?
6. Have you access to a good medical library, or can you obtain books through some incorporated library, from the library of the Surgeon-General?
7. Have you an easy reading knowledge of German or French or both?

Name and Address............................

EXHIBIT B

AMERICAN MEDICAL ASSOCIATION

Section on Ophthalmology

COMMITTEE ON THE COLLECTIVE INVESTIGATION OF QUESTIONS CONCERNING THE OCULAR MUSCLES

183 DELAWARE AVE., BUFFALO, N. Y., January, 1909.

My Dear Doctor:—The committee on the investigation of questions concerning the ocular muscles wishes to acknowledge your reply, and that of many others, offering to cooperate in the proposed study.

The nature of these replies, with other considerations, have convinced the committee that it would be worse than useless to attempt to deal with phases of pathology or treatment, until we can first agree on certain propositions, still doubtful, concerning anatomy and physiology.

The following general plan has therefore been adopted, for the beginning:

1. A given question will be placed, if possible, in the hands of some colleague who has already studied it, or who has expressed a desire to study it.

2. Each colleague is expected to obtain cooperation from others, physiologists, chemists—any one, anywhere—only being certain that apparent facts are really facts, accurately observed and carefully recorded.

3. In physiologic experiments the term "normal eyes" will be understood as those whose manifest ametropia does not exceed 0.05 D., whose proximate point is in proportion to the age, the individual being in good health and entirely free from asthenopic symptoms.

Experience has shown that it is most convenient for a student of these questions to use his own eyes for experiment, if they are sufficiently near the normal type; if not, the eyes of some member of his household. Typical subjects for study can usually be found among soldiers (including members of the National Guard), or others who lead an out-of-door life.

4. For the sake of uniformity each subcommittee or individual is expected to report to the central committee, if requested to do so, two or three times a year concerning the progress made.

5. As accuracy and truth are the prime considerations, it is not desirable to hurry to conclusions. But if a given piece of work in the hands of a subcommittee or of an individual lags unnecessarily, some one else will be invited to study that question.

6. A colleague can feel free to publish his work anywhere and at any time. But if it is to form a part of any report formulated by this central committee, then

A. The article must be in the hands of this committee at least four months before a meeting of this Section.

B. If it has been already published, only a brief abstract would be made of it—sufficient to show its value as a link in the chain of facts.

C. If it has not been already published, an abstract will also be made, but the communication, with abbreviations, if necessary, would be referred to the committee on publication, in order that it may form part of the Transactions of the Section.

7. The central committee, if requested to do so, will furnish references to the bibliography of the questions proposed by them.

If you wish to join in this investigation will you please inform the chairman of this committee before the — of January whether:

A. You will accept the suggestion to study the question given below; or

B. If you prefer some other question, please state what it is.

The central committee appreciates that many who promptly accepted the invitation expecting to make clinical observations, will not care to study first anatomy or physiology. The cooperation of such colleagues must therefore be deferred or unfortunately lost entirely.

But the favorable replies to the first circular letter have been sufficiently numerous, and from men of sufficient standing, to warrant the hope that those who do cooperate will be well repaid by the satisfaction of contributing a little to our knowledge, and by the honor of being connected with a work which may ultimately prove of some credit to American ophthalmology. Truly yours,

THE COMMITTEE,
LUCIEN HOWE, Chairman.

MEMORANDA

MEMORANDA

THE VISUAL FIELDS IN HYSTERIA

A CLINICAL STUDY OF FIFTY CASES*

WALTER R. PARKER, M.D.
Professor of Ophthalmology, University of Michigan
ANN ARBOR, MICH.

The majority of systematic writers on hysteria assert that the most common ocular stigma of this neurosis is a concentrically contracted field of vision. From time to time, however, suggestions have been made intimating the possibility of error in this statement. As my experience seems rather to agree with the minority, I determined to tabulate a relatively small number of cases, the records of which were complete, and present conclusions from this series alone. The results here obtained, however, agree with the findings in a much larger number of cases, the records of which are too incomplete for publication.

Whether the lesion in hysteria be central or peripheral, whether due to anesthesia of the retina, or entirely a matter of suggestion, is not to be considered in this paper.

The cases are all from the neurological or ophthalmological clinic in the University of Michigan. Dr. C. D. Camp, clinical professor of neurology, made all the examinations except those of the eyes, which were made in the ophthalmologic department. All cases in which there were errors of refraction or pathologic lesions that could in any way interfere with the fields were eliminated; otherwise the cases were taken consecutively, and in no way are they selected cases. The fields were all taken on the same perimeter and under like conditions.

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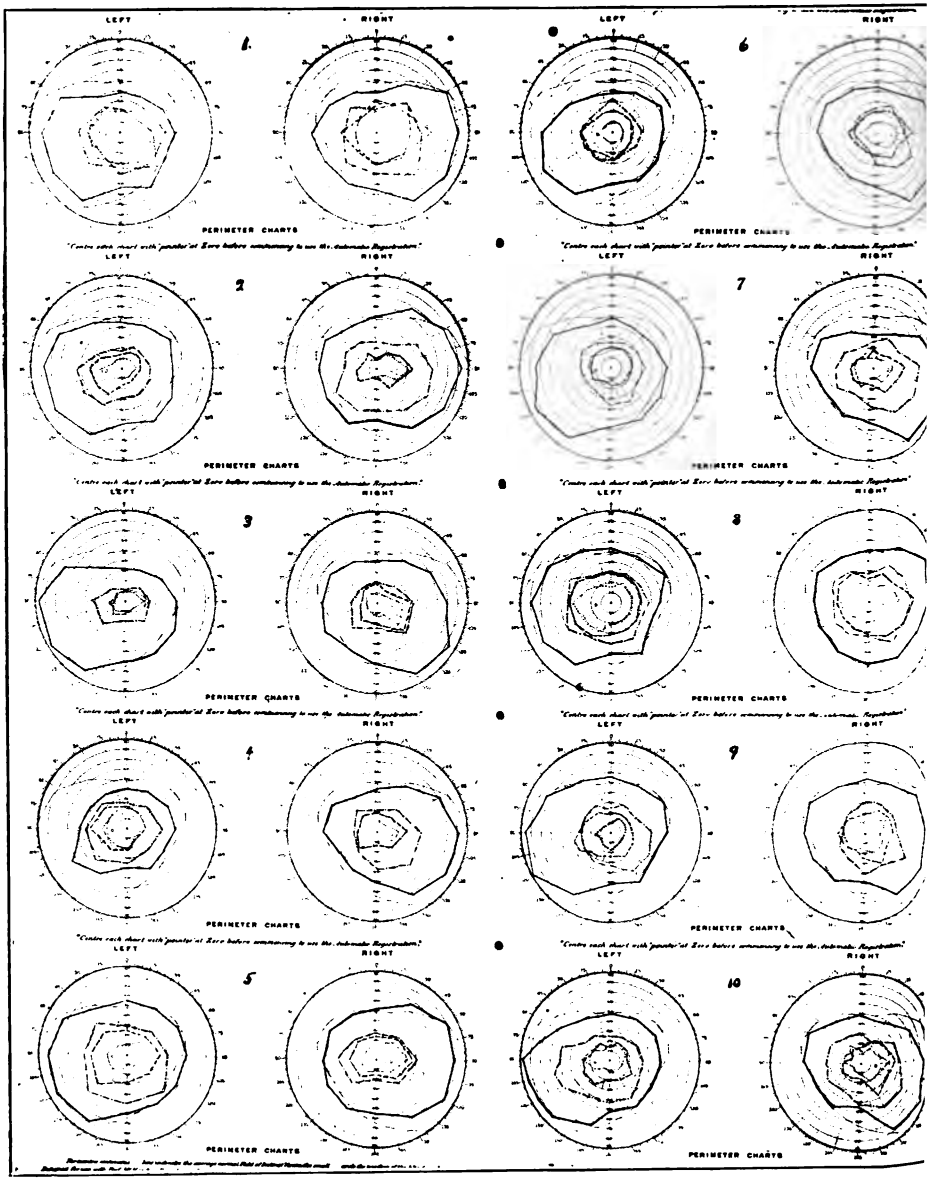
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PERIMETER CHARTS
"Centre each chart with pointer at Zero before commencing to use the Automatic Registration."

Fig. 1.—Visual fields in Cases 1-10. ...... indicates green field. ——— indicates red fiel
—.—.—. indicates blue field. ———— indicates form field.

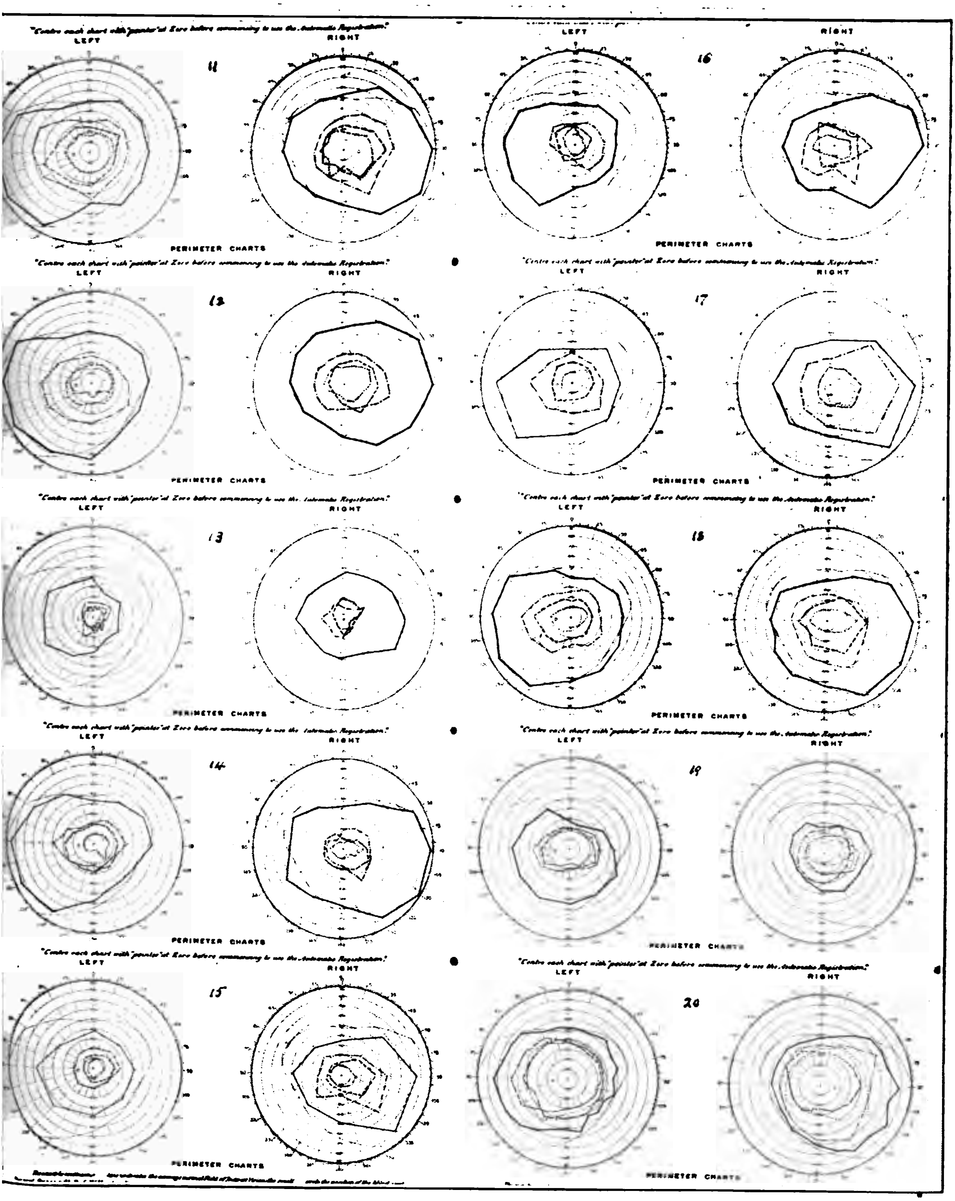

Fig. 2.—Visual fields in Cases 11-20.

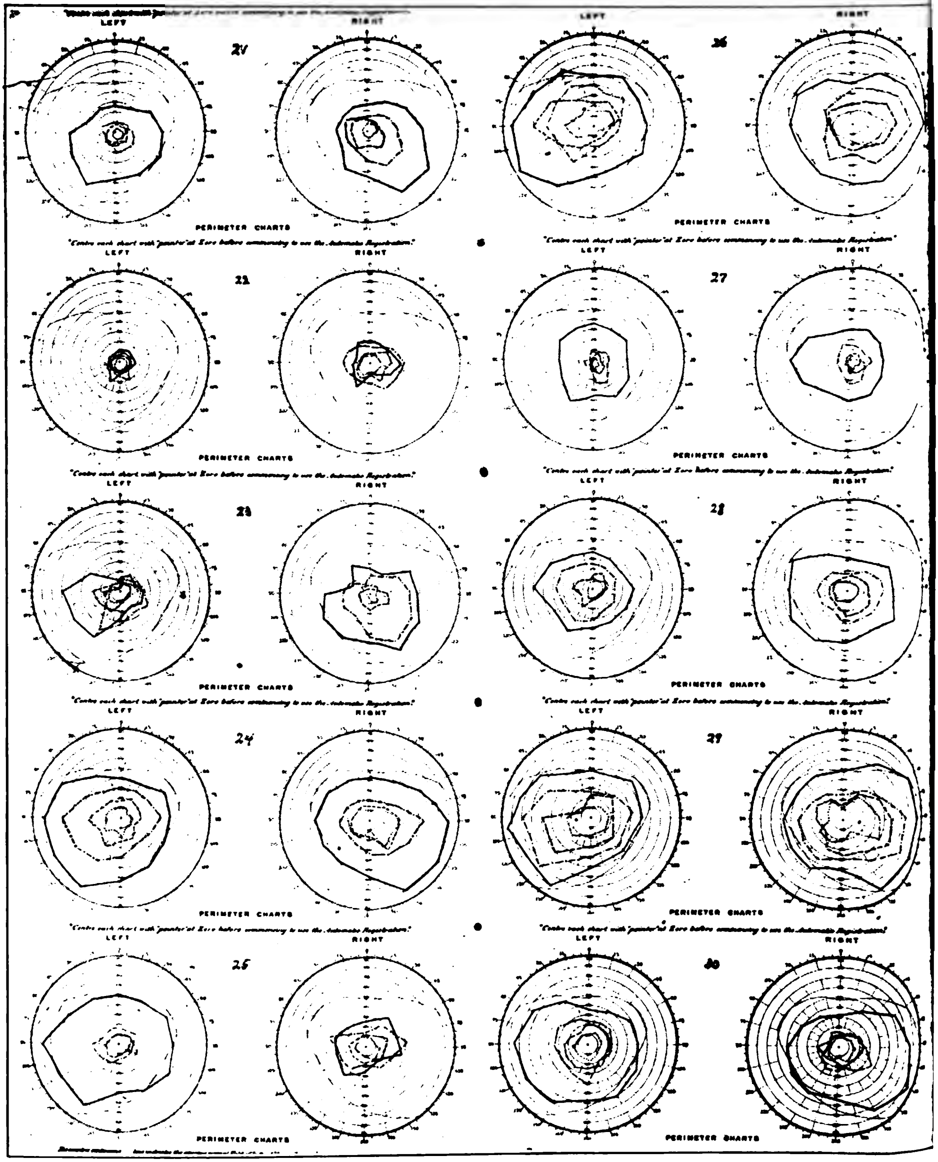
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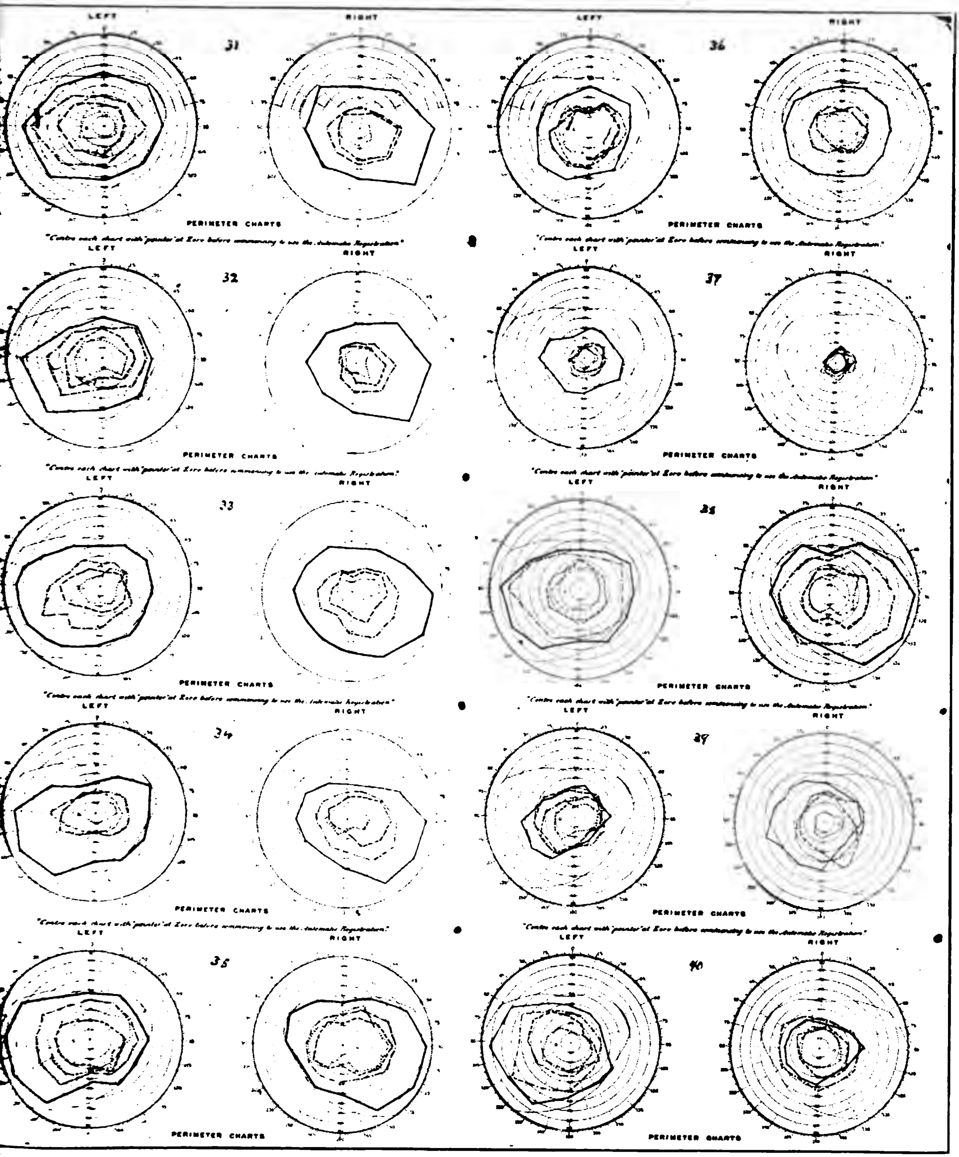
Fig. 3.—Visual fields in Cases 21-30.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PERIMETER CHARTS
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Fig. 4.—Visual fields in Cases 31-40.

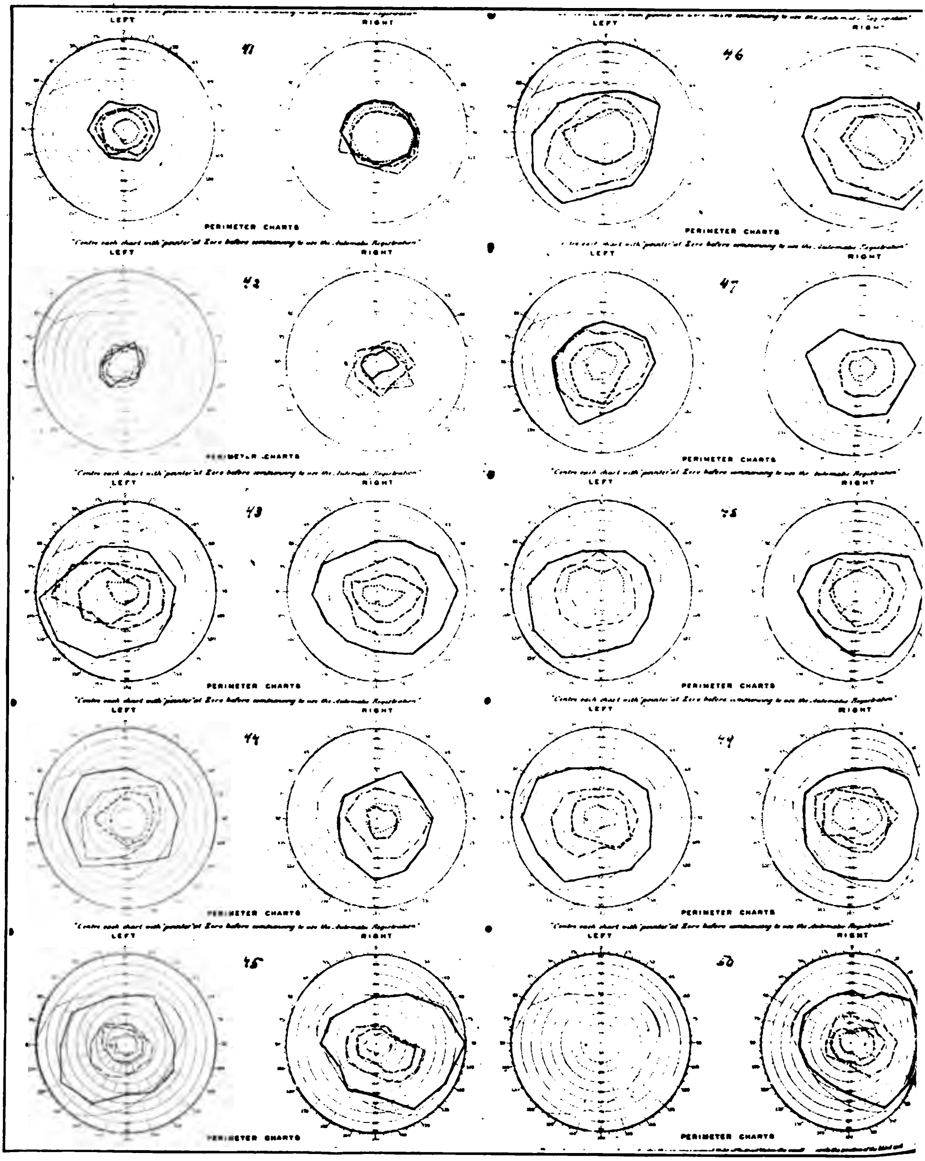

Fig. 5.—Visual fields in Cases 41-50.

Von Graefe suggested that the concentric limitation of the visual fields was the most important ocular sign of hysteria, while dyschromatopsia was first clearly described by Galezowski in 1886. Parinaud says, "The essential sign of hysteria is the concentric contraction of the field of vision, to which may be added in certain cases a particular form of dyschromatopsia." He also thought that the contracted fields were associated with amblyopia. Describing dyschromatopsia, he says, "The narrowing of the visual field, which explains the insensibility to white light, constitutes the fundamental fact of hysterical amblyopia. The disorder of vision as regards colors is a superadded fact which is not constant." In speaking of the field Parinaud says, "It represents an insensibility of a portion of the field to white light, and this insensibility develops from the circumference to the center in such a way that a graphic drawing of the contraction would be represented by an almost circular line." Binswanger, in speaking of the views of the French school which holds that the concentric narrowing of the visual field is the most frequent ocular stigma in hysterical subjects, says, "The skepticism with which we regard the statements of Charcot's school, Galezowski, Parinaud, Landolt, Féré, and others, increases in direct proportion to the growth of our experiences on the subject; for these investigators attribute too much importance to the concentric contraction of the visual field in hysteria." In this country de Schweinitz, in speaking of the concentrically contracted fields, says, "The value of this symptom seems exceeding great, although J. K. Mitchell and myself were unable to find it as frequently among hysterics whom we examined as would appear to be the case in the French clinics." Hansell and Sweet, speaking of the fields, say, "The predominant peculiarity of an hysterical contraction of the visual fields is that, while in every other disease where peripheral limitations occur the color field is affected in equal or greater proportion than the field for white, in hysterical amblyopia the field is less affected for colors than is the field for white. The field for red is apt to be less affected than the field for blue. This reversal of the order and extent of the field for white and colors is positive evidence of hysteria; it can not be simulated and it is caused by no other ocular disorder. It is readily determined and

TABLE 1.—TABULAR VIEW OF OCULAR AND

No.	Sex.	Age.	Eye Symptoms. 1. Dyschromatopsia. 2. Contracted field. (a) Concentric. (b) General. 3. Amblyopia. 4. Scotomata. 5. Intermittent field. 6. Monocular diplopia. 7. Tubular vision.	Other Special Sense Symptoms. 1. Hearing: Hys. deafness. 2. Smell: Anosmia. 3. Taste: Partial loss. 4. Speech: Aphonia.	Sensory Symptoms. 1. Anesthesia of conjunctiva. 2. Anesthesias. 3. Graves' sign. 4. Allochiria. 5. Hyperesthesias. 6. Paresthesias. 7. Hypalgesias. 8. Hyperalgesias.
1	F	15	1, 2-b, 3	....	2, 3
2	F	25	2-b	....	2, 5
3	F	16	1, 2-b	....	1, 2
4	M	..	2-a	....	1, 5
5	F	28	2-b	....	1, 2
6	F	43	1, 2-a	....	1, 2, 5
7	F	16	1, 2-b	....	1, 2, 3, 5
8	F	21	2-b	....	2, 3, 5
9	F	53	1, 2-b	....	1, 2 5
10	F	40	1, 2-b	....	1
11	F	29	1	....	1, 2, 3, 5
12	F	41	1	....	3, 7
13	F	47	1, 2-a, 3	....	6, 8
14	F	16	1	....	1
15	F	37	2-a	....	1, 2, 7, 8
16	F	35	1, 2-b	3	2, 3, 7, 8
17	F	17	2-b	....	2, 3, 7, 8
18	M	19	2-b	....	1, 6
19	F	51	1, 2-a	....	6, 8
20	F	16	1, 2-a	....	....
21	F	32	1, 2-a	....	....
22	F	17	1, 2-a, 3	....	2, 8
23	F	14	1, 2-a	....	5, 8
24	F	..	1, 2-b, 6	4	1, 3, 8
25	F	48	o. s. 1, 2-b, o. d. 2-a	....	2, 6
26	F	33	1, 2-b	....	8
27	F	23	1, 2-a	4	1, 2, 3, 5, 7, 8
28	F	21	2-a, 7	....	8
29	F	25	1, 2-b	....	1, 2, 3, 7
30	F	33	1, 2-b	....	1
31	F	59	2-b	....	6
32	F	16	1, 2-b	....	1, 2, 7
33	F	48	1, 2-b	....	....
34	F	..	1, 2-b	....	6
35	F	13	1	....	8
36	M	29	1, 2-a	....	2, 5
37	F	53	1, 2-a	....	....
38	F	..	1, 2-b	....	6, 8
39	F	53	1, 2-a	....	6, 8
40	M	54	1, 2-a	....	7, 8
41	F	35	1, 2-a	....	6, 7, 8
42	F	39	1, 2-a	....	8
43	F	38	2-b	....	6, 7, 8
44	F	10	1, 2-a	....	1
45	F	46	2-b	....	3, 5, 6, 8
46	F	..	1. 2-b	....	1, 6
47	F	30	2-a	....	8
48	M	19	1, 2-b	....	1
49	F	19	1, 2-b, 3	....	1, 2, 5, 7, 8
50	F	18	2-b	....	5

OTHER SYMPTOMS IN FIFTY CASES OF HYSTERIA

-Motor Symptoms. 1. Paralyses. 2. Tremor. 3. Contractures. 4. Incoordination.	Paroxysmal Attacks. 1. Clavus. 2. Globus hystericus. 3. Epileptoid attacks.	Loss of Reflexes. 1. Absence of pharyngeal reflex. 2. Absence of plantar reflex.	Visceral Symptoms. 1. Vomiting. 2. Vascular and vasomotor disturbances. 3. Rapid respiration. 4. Cardiac disturbances. 5. Cough. 6. Yawning. 7. Phantom tumors. 8. Anuria.	Psychic Symptoms. 1. Hysterical amnesia. 2. Suggestibility. 3. Feeling of unreality. 4. Hallucinations. 5. Hysterical trance.
....	....	....	....	....
....	2, 3	1, 2	....	....
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....	....	2	3	....
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....	....	1, 2	....	....
....	....	....	1	....
....	1	2	4	1
....	....	....	4	....
....	3	....	1	....
....	3	....	....	....
....	1, 3	1	....	....
....	....	....	....	....
....	....	....	....	....
....	2	2	4	....
....	....	....	3, 4	2
1	3	....	....	....
2	1	....	....	....
1	3	....	1	....
....	....	....	....	....
....	1, 3	....	....	1, 4
....	3	....	....	....
....	1	....	8	....
1, 2	....	....	....	1
1	....	1, 2	....	....
....	1	1	....	....
....	1, 3	....	....	....
2	....	....	....	....
....	....	....	....	....
....	....	2	....	....
....	1	....	4	....
....	....	....	....	2, 5
....	1, 2	....	....	....
....	3	1, 2	....	....

TABLE 1.—TABULAR VIEW OF OCULAR AND

No.	Sex.	Age.	Eye Symptoms. 1. Dyschromatopsia. 2. Contracted field. (a) Concentric. (b) General. 3. Amblyopia. 4. Scotomata. 5. Intermittent field. 6. Monocular diplopia. 7. Tubular vision.	Other Special Sense Symptoms. 1. Hearing: Hys. deafness. 2. Smell: Anosmia. 3. Taste: Partial loss. 4. Speech: Aphonia.	Sensory Symptoms. 1. Anesthesia of conjunctiva. 2. Anesthesias. 3. Graves' sign. 4. Allochiria. 5. Hyperesthesias. 6. Paresthesias. 7. Hypalgesias. 8. Hyperalgesias.
1	F	15	1, 2-b. 3	....	2, 3
2	F	25	2-b	....	2, 5
3	F	16	1, 2-b	....	1, 2
4	M	..	2-a	....	1, 5
5	F	28	2-b	....	1, 2
6	F	43	1, 2-a	....	1, 2, 5
7	F	16	1, 2-b	....	1, 2, 3, 5
8	F	21	2-b	....	2, 3, 5
9	F	53	1, 2-b	....	1, 2 5
10	F	40	1, 2-b	....	1
11	F	29	1	....	1, 2, 3, 5
12	F	41	1	....	3, 7
13	F	47	1, 2-a, 3	....	6, 8
14	F	16	1	....	1
15	F	37	2-a	....	1, 2, 7, 8
16	F	35	1, 2-b	3	2, 3, 7, 8
17	F	17	2-b	....	2, 3, 7, 8
18	M	19	2-b	....	1, 6
19	F	51	1, 2-a	....	6, 8
20	F	16	1, 2-a	....	....
21	F	32	1, 2-a	....	....
22	F	17	1, 2-a, 3	....	2, 8
23	F	14	1, 2-a	....	5, 8
24	F	..	1, 2-b, 6	4	1, 3, 8
25	F	48	o. s. 1, 2-b, o. d. 2-a	....	2, 6
26	F	33	1, 2-b	....	8
27	F	23	1, 2-a	4	1, 2, 3, 5, 7, 8
28	F	21	2-a, 7	....	8
29	F	25	1, 2-b	....	1, 2, 3, 7
30	F	33	1, 2-b	....	1
31	F	59	2-b	....	6
32	F	16	1, 2-b	....	1, 2, 7
33	F	48	1, 2-b	....	....
34	F	..	1, 2-b	....	6
35	F	13	1	....	8
36	M	29	1, 2-a	....	2, 5
37	F	53	1, 2-a	....	....
38	F	..	1, 2-b	....	6, 8
39	F	53	1, 2-a	....	6, 8
40	M	54	1, 2-a	....	7, 8
41	F	35	1, 2-a	....	6, 7, 8
42	F	39	1, 2-a	....	8
43	F	38	2-b	....	6, 7, 8
44	F	10	1, 2-a	....	1
45	F	46	2-b	....	3, 5, 6, 8
46	F	..	1. 2-b	....	1, 6
47	F	30	2-a	....	8
48	M	19	1, 2-b	....	1
49	F	19	1, 2-b, 3	....	1, 2, 5, 7, 8
50	F	18	2-b	....	5

THER SYMPTOMS IN FIFTY CASES OF HYSTERIA

Motor Symptoms. 1. Paralyses. 2. Tremor. 3. Contractures. 4. Incoordination.	Paroxysmal Attacks. 1. Clavus. 2. Globus hystericus. 3. Epileptoid attacks.	Loss of Reflexes. 1. Absence of pharyngeal reflex. 2. Absence of plantar reflex.	Visceral Symptoms. 1. Vomiting. 2. Vascular and vasomotor disturbances. 3. Rapid respiration. 4. Cardiac disturbances. 5. Cough. 6. Yawning. 7. Phantom tumors. 8. Anuria.	Psychic Symptoms. 1. Hysterical amnesia. 2. Suggestibility. 3. Feeling of unreality. 4. Hallucinations. 5. Hysterical trance.
....	....	....	....	....
....	2, 3	1, 2	....	....
....	3	1	....	....
....	....	....	....	....
....	....	2	....	....
....	....	....	....	....
....	....	1	....	....
....	....	....	....	....
....	....	....	....	....
....	....	1	....	....
....	....	1	....	....
....	....	....	....	....
....	....	....	....	....
....	....	2	....	....
....	....	2	....	....
....	....	....	....	....
....	....	....	....	....
2	3	....	....	....
....	....	....	....	....
....	....	2	3	....
....	....	1	....	....
....	....	1, 2	....	....
....	....	....	1	....
....	1	2	4	1
....	....	....	4	....
....	3	....	1	....
....	3	....	....	....
....	1, 3	1	....	....
....	....	....	....	....
....	....	....	....	....
....	2	2	4	....
....	....	....	3, 4	2
1	3	....	....	....
2	1	....	....	....
1	3	....	1	....
....	....	....	....	....
....	1, 3	....	....	1, 4
....	3	....	....	....
....	1	....	8	....
1, 2	....	....	....	1
1	....	1, 2	....	....
....	1	1	....	....
....	1, 3	....	....	....
2	....	....	....	....
....	....	....	....	....
....	....	2	....	....
....	1	....	4	....
....	....	....	....	2, 5
....	1, 2	....	....	....
....	3	1, 2	....	....

possesses a diagnostic value equal to the anesthesia of the glottis and pharnyx so frequently referred to as pathognomonic of this neurosis."

The amount of the reduction in the visual fields varies from slight contraction to almost the fixation point. As was suggested by Janet, the amount of contraction has no direct relation to the acuity of vision, all hysterics being able to go about their regular life regardless of the limitation in the fields.

In Table 1 the stigmata at the head of each column are numbered and after each case is placed the numbers representing the stigmata found. A photograph of the field in each case is also attached.

An analysis of the fifty cases under discussion gives the result shown in Table 2.

TABLE 2.—STIGMATA FOUND IN FIFTY CASES OF HYSTERIA

Character of Field.	Stigmata Found. No.	%
1. Dyschromatopsia	36	72
2. Dyschromatopsia with general contraction	17	34
3. Dyschromatopsia with concentric contraction	15	30
4. Contraction of fields without dyschromatopsia	13	26
5. General contraction without dyschromatopsia	9	18
6. Concentric contraction without dyschromatopsia	4	8
7. Concentric contraction, irrespective of dyschromatopsia	20	40
8. General contraction, irrespective of dyschromatopsia	27	54
9. Amblyopia	4	8

CONCLUSIONS:

1. Dyschromatopsia is more common than concentrically contracted fields in the ratio of 72 to 40.

2. There is no relation between the contraction of the visual fields and amblyopia, the former being present in 96 per cent. of the cases, while the latter was found in 8 per cent.

3. There is no constant relation between the areas of anesthesia and concentrically contracted fields. Of the patients with anesthesia 28.4 per cent. showed concentrically contracted fields, while 75 per cent. showed dyschromatopsia.

NOTE.—Not a single case of hysterical hemianopsia has been recorded in either the ophthalmologic or neurologic clinics of the University of Michigan.

The suggestibility of the patients was noted in but few of the cases; otherwise the records were complete.

I wish to acknowledge the assistance rendered me by Dr. George M. Waldeck in the compilation of these cases.

MEMORANDA

MEMORANDA

REGENERATION OF THE CORNEA[*]

MEYER WIENER, M.D.

ST. LOUIS

It has long been known that the epithelium of the cornea would regenerate itself after being destroyed intentionally or by accident.

Haller[1] as early as 1763, in his "Elementary Physiology of the Human Body," asserts that regeneration of the cornea takes place, and even the oldest ophthalmologists[2] assume that regeneration of corneal tissue occurs. In fact, Desmarres[3] states that, in experimenting on transplantation of the cornea, he has noted an increase in length of the corneal flap, and even a true regeneration. Mauchart,[4] Mead and Larry,[4] Wardrop,[4] Gulz[5], and Malgaigne[6] resected corneal scars, and found that healing took place with little or no scar. What is extremely interesting in the light of our present knowledge of the effect of an opium derivative, dionin, is the work of Muehlbauer[7] and Beger,[8] who resected the superficial layers of the cornea and succeeded in obtaining a clear cornea following the application of laudanum.

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[*] This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

[1] "Vulnerato ex succo effuso pellucida renascitur, ut coram video, et late deleta nova redit, omnino ut epidermis." Haller: Elementa physiologiæ corporis humani, Lausanne, 1763, 363.

[2] Mauchart, B. D.: De ungue oculi pure inter lamellas corneæ (defendet C. F. Bilger), Diss. Med., Tübingen, 1742; in Hallerus, Alb., Disputationes chir. selectæ, i, 394, Lausanne, 1755; abstr. in Donders, Note 6.

[3] Desmarres: Ann. d'ocul., 1843, x, 5.

[4] Donders, F. C.: Untersuchungen über die Regeneration der Hornhaut, Holländ. Beitr. zu d. Anat. u. Physiol. Wissensch., 1846-8, i, 387 to 400.

[5] Gulz: Oesterr. med. Wchnschr., 1842, No. 2.

[6] Ann. d'ocul., Fl. Cuniere, 1845, xiii, 211.

[7] Himley: Die Krankheiten und Missbildungen des menchlichen Auges und deren Heilung, 1843, ii, 56.

[8] Ueber die Verwundbarkeit des Auges und seiner Haute, nach Versuchen an Thieraugen, Ztschr. f. d. Ophth. (von Ammon's), iii, Nos. 2 and 4

Desmarres was unsuccessful in his attempt to verify these results, since the rabbits on which he operated contracted a severe purulent inflammation (infection?). resulting in heavy scars.

It is a matter of common observation that corneal wounds following cataract extractions heal so that it would be difficult to find the line of incision. Also numerous corneal opacities clear in time, where the question might easily arise as to whether there might not be a true regeneration of transparent corneal tissue in these cases.

The first systematic research for actual proof of corneal regeneration was made by F. C. Donders[4] in 1846, who suspected that it occurred, but did not think that there was sufficient proof to establish the fact. He accordingly conducted a series of experiments on twenty-two rabbit eyes, from which he resected a flap of cornea from one-third to two-thirds of its thickness, including generally about one-half of the area of the cornea or more. He examined six eyes microscopically and gives a tabulated report showing (1) the thickness of the cornea cut away, (2) the thickness of the uncut cornea, (3.) the thickness of the operated part a definite period after operation, and (4) the thickness of the regenerated part in hundredths of millimeters.

He concludes that the epithelium on the injured part grows thicker than on the normal cornea; that the newly formed true corneal tissue never reaches the same thickness as that removed, but that the longer the time elapsed from the time of operation to the time of examination, the thicker it becomes; that when one-half to two-thirds of the thickness of the cornea is removed. neither inflammation nor any other abnormality occurs; and that epithelium covers the exposed surface within two days after the operation, with a gradual regeneration of cornea, slowly assuming its normal characteristics.

The only one following Donders, so far as I have been able to find, who conducted experiments on animals and made microscopic examinations of the regenerated cornea was Reich.[9] He operated on thirty-two rabbit eyes and made twenty-two microscopic examinations with results similar to those of Donders. The longest time between operation and examination was nine months. His conclusions are that the reaction and the remaining scar are so slight that the removal of thick corneal opacities by the knife are not contraindicated, but that almost always irregular astigmatism and marked light diffusion results.

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[9] Reich, M.: Ueber die Regeneration der Hornhaut. Klin. Monatsbl. f. Augenh., 1873, xi, 197-211.

Waldeyer[10] says that Sanftleven, pupil of Cohnheim, admits the possibility of regeneration of the corneal cells.

The few clinically reported cases of regeneration, all except one, followed destruction of the corneal tissues from purulent ophthalmia. Kaempf[11] reports a case of pyorrhea in a soldier in whom, after the swelling of the lids had sufficiently subsided to permit an examination of the eyeball, an apparent complete destruction of the cornea was revealed, the surface being covered with slough. Within three weeks there was a regeneration of clear cornea, except in the central part, where there was an anterior synechia in both eyes. Four months later an iridectomy performed above and outward in either eye gave useful vision.

N. D. Staicovici[12] reports a case of complete regeneration of the cornea after its destruction down to Descemet's membrane following suppurative conjunctivitis.

A case is reported from Darier's clinic[11] in a man 67 years of age who had been blind for ten years with complete destruction of the cornea with a central prolapse of the iris. The prolapsed iris underwent an inflammation from infection during which the entire cornea sloughed off, leaving only Descemet's membrane, a final regeneration of clear cornea taking place.

H. Armaignac[13] observed, in a patient suffering from a very severe acute purulent coryza, a complete destruction of the cornea with the exception of Descemet's membrane. There was an almost complete staphyloma of the cornea with a central perforation. When the patient went home there was a ring of redness surrounding the cornea two millimeters wide composed of small capillaries which had the appearance of a new growth. The patient was given a 1 per cent. solution of picric acid to instill into the eye. Armaignac was astounded when, a month later, the patient returned to him with a complete regeneration of clear cornea. True, it was traversed by very fine capillaries and sprinkled with small vague opaque spots. The anterior chamber was re-established without synechia. Unfortunately, oblique illumination revealed the pupil almost blocked by a membranous exudate. The patient could distinguish the movement of fingers at thirty centimeters. Atropin failed to break up the adhesions, but one month later vision had reached one-twentieth. The same author[14] reported a case of regeneration of the cornea after a complete destruction of the membrane following purulent ophthalmia in a woman 60 years of age.

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[10] In de Wecker and Landolt: Traité complète d'ophthalmologie, ii, 35.

[11] Regeneration des grossten Theils der Hornhaute nach Pyorrhoe, Oesterr. Ztschr. f. pract. Heilk., 1868, xlv, 77-80.

[12] Staicovici, N. D.: Doua cazuri de regenerere complecta a cornee i in urma destructiunei complecta a acestei memb. membrane, prin. procese de supurantiune conjunctivala, Rev. de chir., Bucharest, 1903, vii; 20.

[13] Armaignac, H.: Un cas de régénération complète de la cornnée aprés un splacele total de cette membrane consécutif à une conjunctivite purpulente chez une femme de 60 ans, Bull, et mém Soc. Franc. d'opth, 1899, xvii, 287.

Peter A. Callan[15] saw the cornea regenerate after its destruction from ulcus serpens. The patient, a boy 17 years of age, appeared at the New York Eye and Ear Infirmary, Oct. 6, 1903, twenty-four hours after having been struck in the eye by a rivet. There was a circular ulcer three mm. to four mm., involving all the layers, filled with a creamy white mass, umbilicated in the center. Despite cauterization and other treatment, the ulcer increased in size until on October 11 the entire cornea was involved and on October 12 the anterior chamber abolished. The next day the slough disappeared, leaving Descemet's membrane exposed. October 14 there was no more suppuration and the eye was bandaged. On October 16 cicatrization was observed over the intact Descemet's membrane. Dionin in increasing strength was given, when by March the upper half had cleared so that the pupil was visible. On June 13 vision with + 3.5 D. cyl. ax. 105 degrees was 8/70. The cornea was quite clear in the upper third, but lower two-thirds more or less hazy, resembling an eye after recovery from a severe attack of interstitial keratitis.

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[14] Armaignac, H.: Regenerazione del tessuto cheratico, Boll. d'ocul., Firenze, 1897, xix, 161-164.

[15] Regeneration of cornea. Report of case of ulcus serpens, Am. Med., July 9, 1904.

H. L. Wolfner[16] reported a case in which a woman 32 years of age came with gonorrheal ophthalmia of right eye. The left eye had been removed in childhood. The socket of the left eye was also infected with gonococci. When first seen there was ulceration of the right cornea, which progressed until the entire cornea down to Descemet's membrane was destroyed. Neither Wolfner nor Dr. A. Alt, who saw the case in consultation, believed that there was any hope of preserving vision. Within six weeks to two months the cornea was practically regenerated. There resulted a very thin but clear cornea, except from a faint cloud just below the center, with a slight tendency toward keratoconus and a small deposit on the anterior capsule; vision, 14/120.

In view of the partial success of the results already obtained along this line, and encouraged by the knowledge accumulated by modern studies on regeneration in general, as well as by the fact that our former investigators had no knowledge of septic surgery, late in July, 1908, I began a series of experiments on rabbits. From then to March 10 thirty-one rabbit eyes have been used for experimentation. Five rabbits died before notations were finished, and these have been excluded from the report. The rabbits were cocainized and wrapped in a long towel, with only their heads projecting, which served to keep them immovable. A thread was then passed through the conjunctiva and subconjunctival tissue so as to help fix the globe, which was held by the assistant who separated the lids. A flap of cornea was then dissected away varying from one-half to a little over two-thirds the thickness of the cornea, and including one-half, two-thirds, or even the entire area of the cornea, the extent of surface bared seeming to have little or no influence on the general healing process or regeneration. The only difference was that the smaller area was sooner covered by epithelium. Aside from taking general aseptic precautions at the time of the operation, no special means were taken for their after-care, the rabbits being placed in the cages without dressing or bandage. Only where occasionally there seemed to be a purulent or mucopurulent discharge after a day or two, a little powdered xeroform was dusted once daily over the cornea until the infection was controlled, which was usually within a few days. Also wherever there happened to be an unusual amount of secretion, purulent or otherwise, which might tend to agglutinate the lids, the lids were washed with warm water and the secretion removed. Following the operation the rabbits seemed to suffer no pain or inconvenience, the palpebral opening being held as wide open over the eye operated on as the sound eye, with no photophobia.

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[16] Regeneration of the Cornea; report of case at Ophthalmic Section of St. Louis Medical Society, June 10, 1908.

Where only half the cornea was removed, the posterior was selected as being more readily managed on account of escaping the incidental action of the membrana nictitans. The section was commenced by making a vertical incision from above downward through the median line from the upper corneal margin to the lower corneal margin, going as deep as judgment from experience would permit without puncturing Descemet's membrane. Then from the center of this line a similar cut was made horizontally backward to the posterior limbus. The triangular was then grasped with a fine conjunctival forceps, and with slight tension, and the knife held flat, the corneal tissue was easily peeled off. Sometimes where the flap looked thin a further attempt to resect another layer was made, but this usually ended disastrously by puncturing Descemet's membrane. When this occurred an ectasia corneæ, or even staphyloma of the cornea, almost invariably took place. Twenty-four hours after the operation there was usually some slight conjunctival injection. The resected surface had a stippled appearance as though covered with a thin layer of serum or lymph, but wiping the surface with a wet sponge did not seem to change its aspect. The cut edges had a slightly raised but rounded-off look. The transparency, however, was surprisingly little disturbed. This was also the case after forty-eight hours, when the raised edges had encroached several millimeters over the raw surface. There were at this time, however, no blood vessels over the cornea except that there was an increased vascularization at the limbus.

On the third day there is generally a small milky spot about the center of the resected area, but this usually disappears within a few days, when the cornea again shows a smooth surface, being completely covered by epithelium. The resected area now shows a general infiltration or haziness which is greatest at the line of incision, but at no time to such an extent that the pupil can not be seen throught it, unless infection supervenes. This haze gradually clears until about four to six weeks after the operation a casual examination would not determine an opacity, which can only be revealed in certain positions or by oblique illumination.

In those rabbits which have been kept several months the cornea is generally apparently perfectly clear by oblique illumination as well as by transillumination, with the exception of an occasionally small, slightly hazy area.

Microscopic serial sections have been made showing the fresh cornea immediately after resection; after twenty-four hours; after forty-eight hours; four days; seven days; two weeks; one month; two months, and so on up to seven months. The specimens show a gradual thickening of the cornea the longer the period from the time of operation. Experiments are also under way on other animals than rabbits. Since, however, enough time has not been had to complete the series, I have refrained from presenting at this time the results of microscopic examination, not wishing to draw too hasty or indefinite conclusions. This is consequently offered as a preliminary report.

500 Carleton Building.

MEMORANDA

SCISSORS-MAGNET EXTRACTION OF IRON FROM EYEBALL*

EDWARD JACKSON, M.D.
DENVER

Particles of iron or steel may remain embedded deep in the eyeball for long periods without becoming so enclosed or so firmly adherent as to prevent magnet extraction by the ordinary methods. In the lens the development of a traumatic cataract may rather facilitate removal; and particles remain freely suspended in the vitreous for considerable periods, responding to the attraction of the magnet very much as they would immediately after the injury. De Schweinitz[1] has reported cases in which the tip of the Sweet magnet placed at the scleral incision, not introduced within the eye, promptly removed steel from the vitreous in one case in five weeks, and in the other two months after the injury; and in a third case of six months' duration, although the foreign body was not secured on the magnet, subsequent inability to demonstrate it within the eyeball where it had previously been localized by the *x*-ray, and the subsidence of the retino-chorioiditis it had caused, seemed to justify the assumption that it had been removed. Hirschberg,[2] speaking of the Schloesser-Edelmann magnet, says, "With it I once extracted a splinter of 27 milligrams which had been for five months embedded in the retina and had already induced siderosis of the globe. The result was permanent and good."

In the series of Knapp and Stoll[3] a fragment was extracted through a scleral incision, after repeated trials with a giant magnet had failed to bring it forward, in one case at the end of six weeks, in another after 107 days. In the third case, after four months had elapsed, the foreign body was brought forward with a giant magnet and extracted through the cornea. At the Royal London Ophthalmic Hospital, as reported by McCallan,[4] foreign bodies were extracted from the depth of the eye (some of them, however, from the lens) after five weeks, seven weeks, six months (three cases), two years and three years.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

[1] Tr. Am. Acad. Ophth. and Oto-Laryngol., 1904, p. 164.

[2] Ophthalmoscope, February, 1905.

[3] Arch. Ophth., July, 1907.

Operative failures, however, are not so generally reported as successes; and in the literature there is abundant evidence that after a foreign body has been embedded behind the iris and lens for more than one month successful magnet extraction is the exception rather than the rule. McCallan, among 39 cases, most of the patients being treated shortly after injury, gives 4 in which the giant magnet failed to extract the foreign body and indicates definite causes for this failure: "In the first two cases the foreign body was encapsuled and adherent to the ciliary body; in the third case the piece of steel was very large and was firmly lodged in a rent in the sclerotic."

In a case reported by de Schweinitz, although the fragment of steel was large, it could not be drawn from its position in the sclera by a magnet in contact with it. Sweet,[5] among 57 cases of foreign body in the posterior portion of the eyeball, located by the Roentgen rays, records 6 in which the magnet failed to extract; 5 of the eyes were enucleated and a piece of magnetic iron or steel found embedded in fibroplastic cellular tissues. The periods in which this firm fixation of the fragment had occurred were, respectively, eighteen years, three years, ten months, three months, five weeks, twelve days and four days. Millikin,[6] who had to extract such a foreign body with forceps, says: "I do not believe that the magnet has yet been invented that will draw small foreign bodies through tough fibrous tissue."

In the series of Knapp and Stoll,[7] among 9 cases in which the foreign body was recognized in the vitreous with the ophthalmoscope, the giant magnet failed to extract it in 4 of them; and in 5 other cases in which

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4. Royal London Ophth. Hosp. Rep., xv, 156.
5. Tr. Am. Ophth. Soc., 1901, p. 362.
6. Tr. Am. Ophth. Soc., 1892, p. 572.
7. Arch. Ophth., January, 1906.

the foreign body was certainly present, though not visible, there was failure to extract with the magnet. Hirschberg, referring to a series of 64 of his later cases, says: "In 4 cases only of old splinter in the fundus (a small number in my opinion) was I unable to carry out extraction." Haab[8] gives as the reasons for failure in magnet extraction: 1. "The foreign body was seated too firmly in the back wall of the globe or had pierced it completely. 2. The splinter was seated in the ciliary body at first or was drawn there by mistake. 3. The splinter had produced fibrinous-purulent exudation which, according to my experience, greatly hinders its movability. 4. The splinter had been healed over in the course of months or years." Snell[9] says the prospect of saving an eye will be inverse to the interval between the injury and the time when the electro-magnet is employed.

Enough has been said to develop the point that in a considerable proportion of cases the electromagnet, whatever its form and however used, fails to extract pieces of iron capable of magnetic attraction, because they are too firmly embedded in recent exudate or organized tissue. Such cases are sufficiently numerous to make it of interest and importance to consider a method by which some of these eyes may be saved; and such a method I believe we have in the use of scissors held in contact with the magnet while used to set free the entangled foreign body. This is shown by the following cases:

CASE 1.—*Fragment of steel in eye over five months. Beginning siderosis. Failure of giant magnet; localization by x-ray. Failure with large hand magnet. Extraction with scissors. Almost full vision.*

History.—A. N. A., a man, aged 21, was struck in the left eye, Feb. 26, 1907, by a bit of steel flying from a hammer. His fellow workmen said that a stream of blood came from the wound, and that something must have gone into it. (The accident happened in southern Mexico.) The doctor the patient saw could find nothing in the eye. A cloud appeared in the upper part of the field of vision, but this passed away in about two weeks, and the eye was not badly inflamed. The patient could see perfectly after this. The vision became gradually impaired, however, and had grown more rapidly worse for the last month. He now consulted a competent ophthalmologist, who made repeated trials on each of five different days with a very powerful giant magnet. Each trial caused very severe pain in the lower part of the eye so that he felt very nervous the rest of the day; but it produced no bulging of the iris or other displacement of the foreign body.

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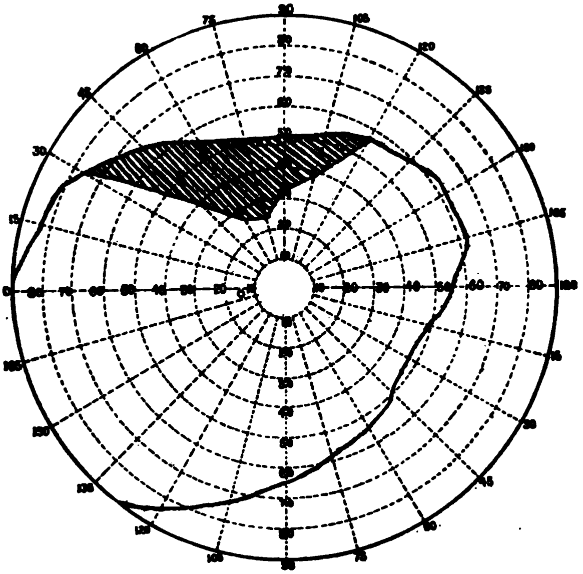
[8] Haab, O.: Removal of Foreign Bodies from the Eye, THE JOURNAL A. M. A., 1902, xxxix, 463.

[9] Ophthalmoscope, February, 1905.

Examination.—August 6, 1907, he consulted me. Vision: R. 4/4 —; L. 2/100. There was a scar 1 mm. long in the nasal margin of the cornea. No opening was visible in the iris; but in the nasal part of the anterior vitreous was a triangular opacity, and the vitreous generally was hazy. Under atropin the opacity in the vitreous looked almost like detached retina, but was devoid of vessels. Radiograms made by Dr. G. H. Stover showed a very distinct shadow, indicating by the localization method of Sweet, a foreign body 9 mm. back of the center of the cornea, 6 mm. to the nasal side of the median plane, and 4.8 mm. below the horizontal plane.

Operation.—August 8, after the free use of cocain in crystal, an incision was made in the conjunctiva below the nasal border of the cornea. Then, pushing this aside so that it would give a valvular opening, a meridional incision was made in the sclera, beginning 7 mm. back from the corneal margin and extending 8 mm; the knife, entering 8 mm. in the direction of the foreign body, came into doubtful contact with it; an attempt being made with it to cut both above and below the particle of iron, with a rather free incision in front of it. The tip of the Johnson magnet was introduced twice, as nearly as possible in contact with the fragment. It was seen to drag the eyeball out of shape. When the particle was thus drawn on an attempt was made to seize it with the forceps, but this was unsuccessful. I then introduced scissors with blunt points as near to the foreign body as possible. Then the round end of the core of the magnet was placed in contact with the joint of the scissors, the current was turned on, and five or six distinct short snips were made with the scissors. They were then withdrawn, still in contact with the magnet, and the foreign body found adhering to the blades. It was the shape of an arrow-head 2.5 by 1.7 by 0.7 of a millimeter in size. The operation caused pain and faintness, especially during the first attempts to extract with the magnet; but the pain was not so bad as had been caused by the attempts to extract with the giant magnet. The eye was closed without any suture.

Postoperative History.—At the end of twenty-four hours there was moderate swelling in the parts, but the lines of incision could not be detected. General hyperemia of the globe was moderate; there was no pain. Vision had risen to 3/60, the patient noticing as soon as the eye was uncovered that "things looked much clearer." The general clouding of the vitreous was noticeably less, and in place of the dense opacity large floating shreds of white tissue were seen. The pupil was circular, 7.5 mm. in diameter. The eye was now left open and treated with atropin boric acid collyrium. The next day the hyperemia was less; a fair view of the fundus was obtained. Vision was 2/45. From this time improvement was steady and rapid. On the fifth day vision 4/20 partly, and on the 30th day 4/9 partly. At the end of three months it was 4/5 partly, at which it remains. The patient has finished a course in engineering with drawing and outside office work, without trouble with his eyes. The ophthalmoscope showed a very faint general haze in lower anterior vitreous, with a white scar having pigmented edges 1 disk diameter long and 1/3 disk diameter wide, at the extreme periphery of the fundus. The field of vision taken ten months after the operation is shown in the acompanying figure.



Field of vision ten months after operation in Case 1.

CASE 2.—*Fragment of steel in the eye three years. Partial traumatic cataract; incipient siderosis. Failure of magnet alone. Scissors-magnet extraction. Preservation of useful vision.*

History.—W. A. H., a man, aged 34, came to me October 18, 1907, at the suggestion of Dr. L. G. Woodson, Birmingham, Ala. Three years previously his left eye had been struck by a small piece of steel. The resulting inflammation lasted about three weeks, but it had not been sore since. During the last two months the vision had been growing worse, although there had been some temporary improvement under treatment.

Examination.—The vision was R. 4/4; L. 4/15. The tension of eyeball was normal. The left iris was discolored, decidedly brownish, as compared with the right. The pupil was a little sluggish as compared with the right, slightly oval, long axis vertical. At the temporal periphery of the iris there was a small coloboma, although the scar in the limbus was not perceptible. The ophthalmoscope showed the right eye normal except that the smaller retinal vessels were slightly irregular in caliber and tortuous. In the left eye there was good fundus reflex in all directions through both the natural and the acquired pupil; but the details of the fundus were not well seen on account of the lens opacity. Under atropin this opacity was found to be net-like in appearance, situated in the deep layers of the posterior cortex, and about the posterior pole of the lens. Through it the larger retinal vessels appeared normal. In the extreme periphery of the fundus, downward and slightly inward, a black mass with a sharp edge projected into the field of view, probably the foreign body. Radiograms, made by Dr. G. H. Stover, gave by Sweet's method of localization, the shadow of a foreign body located 12 mm. back from the center of the cornea, down 9 mm. and to the nasal side of the median plane 4.5 mm., its size appearing to be 2.5 by 0.5 by 0.5 mm.

Operation.—November 20. Under local anesthesia with cocain crystals an incision was made by lifting the conjunctiva on the point of the knife, dragging it toward the vertical meridian, and making the scleral cut, starting 6 mm. back from the corneal margin, and 4 mm. to the nasal side of the vertical meridian, extending from this point 5 mm. back and slightly toward the nose. There was free bleeding from chorioidal vessels. The tip of the Johnson magnet was introduced twice, first between the lips of the wound, and later in contact with the foreign body. Slight pain was produced when the current was turned on, but there was no dislodgment of the foreign body. The tip of the scissors was then introduced as close as possible to the foreign body. The first time, the current was turned on before the magnet was in contact with the scissors, which were consequently jerked out of the wound. They were reintroduced, the magnet was brought in full contact with the joint of the scissors, and the turning on of the current produced no disturbance in the position of the scissors. About four snips were made with the scissors, which were then withdrawn, still in contact with the magnet, bringing the foreign body away on the tip of the blades. The foreign body was found to be a bit of steel 2.25 by 1.5 by 0.6 mm. in size, entirely covered with rust, except one edge. The bleeding ceased promptly; the pupil was widely dilated. There was some smarting in the eye during the day, but no aching.

Postoperative History.—November 22: There had been no pain. There was moderate ecchymosis. With the ophthalmoscope the black mass was gone from the lower periphery of fundus, and a white area was seen in its place. No hemorrhage was visible. Otherwise the ophthalmoscopic picture was unchanged.

November 27: Vision, L. 4/22, appearance of eye normal, except ecchymosis.

The eye remained entirely quiet after that time. At the end of two months, vision had risen to 4/12 + at which it still remains, without noticeable change in the lens opacity.

In both the above cases I had the counsel and assistance of my colleague, Dr. E. W. Stevens.

My first practical acquaintance with magnet extraction was as an assistant in the attempted removal of a minute fragment of steel, suspended in the vitreous a little way back of the lens, where it was easily watched with the ophthalmoscope. The approach of the magnet, or the turning on of the current, caused the fragment to be noticeably attracted toward the magnet; and this attraction increased as the magnet was brought nearer, until the tip, being introduced into the vitreous, began to push the vitreous before it, and so caused the entangled splinter to retreat. The attempt to remove the splinter was repeated several times, and always with the same result. The fragment was each time pushed away before the magnet tip until the foreign body became obscured by hemorrhage, and the eye was finally lost by suppuration. The magnet used was a small one. Its attractive force was not sufficient to overcome the resistance of the tissue in which the foreign body was embedded.

In every case successful magnet extraction depends on the sufficiency of the attractive force to overcome the hold of the tissue on the foreign body. Since the attractive force exerted is proportioned to the size of the particle to be extracted, the smaller particles require the use of a more powerful magnet. The giant magnet has the advantage of exerting its maximum power from a greater distance. But for particles embedded in the coats of the eye, or in blood clots, exudate, or organized scar tissue, no magnet yet made, or likely to be placed at our disposal, is sufficient to overcome the resistance

MEMORANDA

THE ACCOMMODATION AND DONDERS' CURVE AND THE NEED OF REVISING OUR IDEAS REGARDING THEM

AN EXPERIMENTAL STUDY *

ALEXANDER DUANE, M.D.
NEW YORK

NEED OF REVISING DONDERS' WORK

Of the many invaluable contributions that Donders made to our science certainly one of the most important was his determination of the range of accommodation at different ages. His researches, indeed, all admit to be fundamental. For that very reason we must avoid ascribing to them an exactitude and a validity which they do not possess and which Donders himself did not claim for them.[1] For we must remember that the number of cases utilized by Donders was small and the individual variations that they presented were rather considerable. Thus his estimate that 7 D represents the normal range of accommodation at the age of 30 is based on but five cases, the accommodation of which was found to vary between 6 and 9 D. It is obvious that more numerous observations might have extended these limits in either direction and have given a different value for the mean. Furthermore, the observations of Donders were based on the assumption that his subjects were actually emmetropic — an assumption

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

* A preliminary statement of the results here given was presented to the American Ophthalmological Society last summer. This statement, however, being based on insufficient data, was intended to be provisional only, and as a matter of fact contains some inaccuracies.

[1] He says: ,'The deviations [from] the mean course are not particularly great. And still they must undoubtedly in part be ascribed to errors of observation."

which, in the absence of precise cycloplegic tests, must be regarded as erroneous for at least some of them.[2] Finally clinical experience has proved to many of us that in some respects the course of presbyopia runs otherwise than Donders taught.

In view of these facts and of the great practical importance of the subject it seems advisable to extend and check Donders' investigations as fully as possible.

OUR OBSERVATIONS; METHODS EMPLOYED

The observations here detailed, made by Dr. J. B. Thomas and myself, are a contribution in this direction. The plan which we have been pursuing—for our investigations are still unfinished—is to render each patient emmetropic by applying the full correction of his refractive error and then to determine his near point with Prince's rule. This gives the corresponding amount of accommodation in diopters, which, as the patient's far point has been placed at infinity by his glass, evidently represents his full accommodative power.

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In carrying out such a plan the following precautions must be observed:

1. The subject must be made really emmetropic, i. e., his refraction must be wholly and accurately corrected. To effect this in our cases the refraction was carefully determined in every one, and a cycloplegic was used in all but a very few under 48 and in some over that age. Special care was taken to determine the astigmatism accurately to within 0.12 D.

2. The subject's vision must be such that he can make the fine distinctions required. We were, therefore, obliged to reject cases of irregular astigmatism, most cases of high myopia and of high astigmatism even when corrected, of amblyopia from any cause, but especially amblyopia associated with any defect of central vision, of poor sight from corneal or lenticular opacities, etc. Persons thus affected can not discriminate between the blurring due to accommodative fail-

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[2] Thus Donders, speaking of the hyperopia which he believes to develop later in life, says: "I have not infrequently met with this in persons at sixty years of age, who in their youth probably exhibited no H whatever. This was inferred when they did not before the forty-fifth year of their life, need spectacles in the evening for minute work." As up to the age of forty-five a hyperopia of one or even two D may remain latent, such an inference is not to be relied on. Donders himself admits: "In some a slight degree of [latent] H may also increase the deviations."

ure and the blurring due to their naturally defective vision. In general no subject was utilized whose vision was less than 20/20.

3. The full correction being applied, and the eye not under examination being screened, the test object is brought up until it blurs, then withdrawn until it clears, and then carried back and forth several times until we can be sure of the very nearest point where it begins to blur. This will be the near point, and the corresponding amount of accommodation in D, read off from the scale, will give the patient's range.

4. If the near point is closer than 10 cm. a slight error in determining its position will involve an error of one or more D in the value found for the range of accommodation. In such cases, after making the test with the full correction alone, it is well also to make it with this correction combined with a—3.00 or—4.00 D sphere, so as to carry the near point out beyond 10 cm. The value then found for the range must be increased by the amount of this added lens. But it must be noted that the estimate thus obtained is generally too low, and a value found in the ordinary way which does not exceed such estimate by more than one or two D, may be regarded as accurate.

5. If the near point is beyond 40 cm. it must be brought within measurable distance by the addition of a convex glass to the patient's full correction. From the value of the range thus found the strength of this convex addition must, of course, be subtracted. The addition in any case must be as small as possible, for a strong convex glass often brings the near point closer than it should be theoretically, so that the value of the range found with it is too great by a diopter or more. This will not happen if the convex addition is made only just large enough to bring the near point to between 30 and 40 cm.

6. For reasons that will be enlarged on later it is necessary to make repeated tests in the same case in order to discover the subject's accommodative power. This is especially true of young subjects in whom the first test rarely reveals the true range. Because of this fact we have been obliged to exclude a number of observations in which we were able to make the test but once under proper conditions, or in which the subject afterward exhibited a greater accommodation.

7. We must make sure that the patient tested understands just what is required of him. In particular we must tell him (a) to put forth all his efforts and strain as hard as he can, so as to focus down on the test object at the closest possible point; (b) to be careful to indicate the precise point where the test object begins to blur—not the point where it becomes entirely confused.

8. We must be sure that the patient is physically fit for the test. He must not be incapacitated from exerting his full accommodative power by muscular exhaustion, neurasthenia, or similar causes. In our series of cases we had to reject a number of observations in which the patient exhibited a temporary incapacity thus occasioned.

9. The test object must be satisfactory. None has proved perfectly so for all persons and at all ranges. Donders used a wire optometer and fine print. For most people, however, these two tests, and especially the latter, make the near point appear too close — giving thus an overestimate of the range. After trying various objects — dots in pairs and groups, parallel lines, simple geometric figures, etc.—we found the best test object[3] to be a single engraved black line, 0.2 mm. thick and 3 mm. long, bisecting a dull, white, oblong card which is 3 mm. long and 1.5 mm. wide and is mounted on a square of black velvet. The moment the line is brought within the near point it blurs (broadens) and then doubles. The line should be so held as to be most clearly seen. Most people see it best when held vertical.

10. The illumination should be suitable. This is a matter of great importance, and lack of attention to it may cause an underestimate of one or more D. The light must be good but not dazzling (diffused daylight is the best), and must come almost straight from behind — over the right shoulder when the right eye is tested and over the left shoulder when the left eye is tested. There must be no shadow on the card which bears the line and no dazzling reflections nor confusing lights beyond it or alongside. For this reason when the

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[3] Hess recommends Scheiner's test. We have tried this in various modifications, but none has proved satisfactory as a clinical test, that shall be applicable to all sorts of patients, intelligent and otherwise. Moreover, experimenting on myself, I find that the single line, if made fine enough and sharp enough, gives almost precisely the same results as Scheiner's test and is not subject to certain chances of error that inhere in the latter.

test is made with Prince's rule, the latter should be held with its broad side vertical, for if held horizontal the graduations on the rule and the glare from its surface are apt to distract the patient's attention or cause confusion.

11. We must be careful to define the point from which we make our measurement, and take care that we do actually measure from this point each time. Donders measured from the nodal point of the eye, while the tables usually given presuppose a measurement made from the anterior principal point of the eye, i. e., practically from the anterior surface of the cornea. I prefer to measure from the anterior focus of the eye, i. e., from a point 13 mm. in front of the cornea, because this is the point at which we place the convex glass that we use to measure the accommodative power with and to replace the latter when absent. Thus when we speak of a deficiency of accommodation of 6 D we mean one that will be compensated for by a lens of that strength placed at the anterior focus—not at the anterior principal point—of the eye. So, too, when we speak of a hyperopia of 6 D we mean one which is corrected by a convex glass of that strength placed at the anterior focus of the eye. If we shifted the compensating glass to the anterior principal point of the eye we should need one of 6.5 D to accomplish the desired result.

COMPARISON OF OUR RESULTS WITH THOSE OF DONDERS

The values that we found in the range in our cases are plotted in the accompanying diagram (Fig. 1). Each subject is denoted by a separate dot, and the same subject at different ages is denoted by two dots connected by a wavy line. In Figure 2 the broken line, AA, denotes the mean of our observations; the continuous line, BB, the similar curve that Donders constructed from his observations; CC denotes the upper, and DD the lower of the limits between which the great majority of our observations are comprised.

BB, it will be observed, differs from Donders' curve as ordinarily depicted in the text-books in two particulars. In the first place, it rises higher at the start, reaching 18 instead of 14 D. In the second place, it does not touch the base line or O even at the age of 70. The reason for the first difference is that Donders' curve as ordinarily drawn represents the near point when measured from the anterior principal point of the eye, while, in order to make it comparable with our curve, we had to draw it as if measurement had been made from the same point as for the latter, i. e., the anterior focus of the eye. The second point of difference arises from the fact that Donders' curve as usually drawn shows the absolute position of the near point in his patients, whether they were emmetropic or hyperopic, while, to make it comparable with ours and to show the actual range of accommodation as he found it, we had to change the curve to what it would be if his elderly hyperopic patients were rendered emmetropic by glasses. To do this we had to shift the far point in all his hyperopic cases up to the base line and shift the near point correspondingly upward.

It will be noted that our curve differs from that of Donders' in three important particulars:

1. Before the age of 20 our curve falls below his. Here, owing to the comparatively small number of cases and to the difficulty of getting accurate results when the accommodation is high and the patient is young, the results must be regarded as uncertain. Yet we think that the probable error in our findings is not over one D, and that in any case Donders' results for the early ages (10 to 15) are distinctly too high.
2. From 20 to 45 our curve rises above his.
3. From 45 on it falls below his. The difference here is due to the sharp plunge that takes place between 38 and 50.[4]

Both according to our observations and those of Donders the accommodation remains nearly constant after the age of 51, diminishing not more than 0.50 D in ten years. We find a somewhat lower range than Donders did for this time of life.

Hess believes that the accommodation shown in advanced life is in part spurious, the patient being able to see distinctly within his true near point, because his pupil is very narrow and hence has the sharp defining power of a stop or stenopæic aperture. But in a number of patients that we examined between the ages of 50 and 60 the pupil was fully 3 mm. in diameter, so

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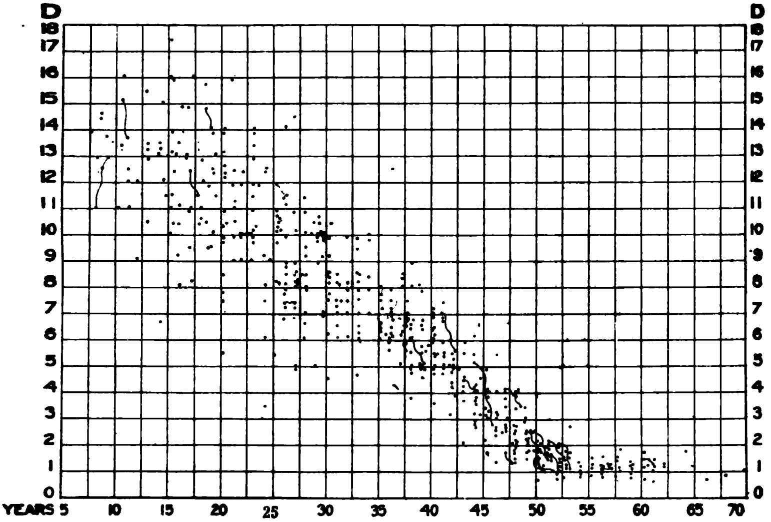
[4] The fall in accommodation between these ages according to our observations is 4.8 D, or just double the amount found by Donders.



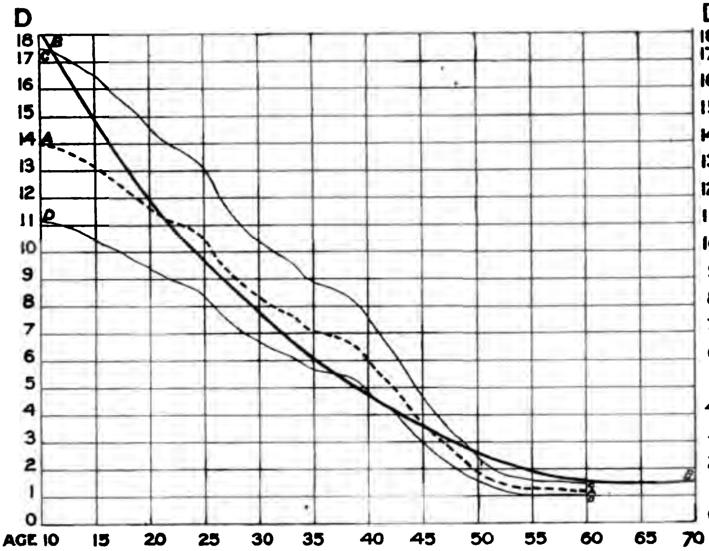


Fig. 1.—Range of accommodation at different ages. Each dot denotes a separate subject, the position of the dot indicating the subject's age and the amount of his accommodation. Two dots connected by a wavy line mean the same subject at different ages. The near point in all cases was measured from the anterior focus of the eye (i. e., from a point 13 mm. in front of the cornea).

Fig. 2.—Comparison of observations with Donder's curve. AA, curve representing the mean of our observations. (Before the age of 25 and especially before the age of 15, this curve can not be regarded as certainly determined, although it is probably not over 1 D out of the way.) BB, Donders' curve, modified so as to conform to our plan of measurement (from the anterior focus of the eye) and to represent the absolute range and not, as is usually done, the absolute near point in his cases. CC, represents the upper and DD the lower limits for normal subjects according to our observations.

that Hess' explanation would not account for the range of 1 D or more that these patients showed.

The numerical differences between Donders' observations and ours is shown in Table 1.

TABLE 1.—NUMERICAL DIFFERENCE BETWEEN DONDERS' OBSERVATIONS AND CONTROL OBSERVATIONS

Range in D, near point being measured from anterior focus of eye = 1.3 cm. in front of the cornea.

Our Observations.					Donders' Observations.	
Age.	No. of Cases.	High.	Low.	Mean.	Mean.	No. of Cases.
10	4	16.0	11.0	14.0*	18.0	5
15	14	17.5	10.0	13.2*	15.0	8
20	16	14.0	6.5†	11.7	11.8	7
25	13	12.0	5.5†	10.5	9.7	4
30	18	10.5	4.5†	8.4	7.8	5
35	9	8.1	6.0†	7.1	6.0	2
40	16	7.3	3.7	6.0	4.8	5
45	13	5.0	1.8†	3.8	3.7	..
50	12	2.6	0.7	1.9	2.6	..
55	4	1.6	0.8	1.3	2.0	..
60	5	1.7	0.7	1.2	1.5	..

* Approximate.
† Probably abnormal.

TABLE 2.—NUMERICAL DIFFERENCE BETWEEN DONDERS' OBSERVATIONS AND CONTROL OBSERVATIONS

Range in D, near point being measured in the ordinary way from the anterior principal point (practically from the anterior surface of the cornea).

Our Observations.		Donders' Observations.	Our Observations.		Donders' Observations.
Age.	Mean.	Mean.	Age.	Mean.	Mean.
10	11.6*	14.0	40	5.5	4.5
15	11.0*	12.0	45	3.6	3.5
20	10.0	10.0	50	1.9	2.5
25	9.1	8.5	55	1.3	2.0
30	7.5	7.0	60	1.3	1.5
35	6.4	5.5	65	1.2	1.5

* Approximate.

NORMAL VARIATIONS IN THE RANGE OF ACCOMMODATION

What chiefly concerns us as clinicians is not so much the mean value of the accommodation at any given age as the limits within which the accommodation at that age must lie in order to be regarded as normal. A study of Figure 1 shows that the great majority of our cases lie between the limits indicated by the lines CC and DD in Figure 2, i. e., have a range of not more than 20 per cent. below or of 25 per cent. above the mean. The numerical values of these limits for each age are given in Table 3. An accommodation which is persistently above the upper limit or persistently below the lower must be regarded as abnormal. But it can not be so regarded on the strength of a single observation, no matter how aberrant the finding. For such a finding is usually erroneous, the error occurring because:

1. Our method of examination is faulty and, in particular, the test object is unsuitable or the illumination is defective.

2. The patient is inattentive or fails to comprehend what is required of him.

3. Through inertia, laziness or fatigue he fails to exert his full accommodative power.

Owing to these sources of error the same patient when examined at short intervals often shows wide variations —variations which, however, do not mean any real change in his accommodative power.

The usual effect of these sources of error is to make us underestimate the patient's accommodation. Provided we can be sure that the proper conditions are fulfilled, the highest measurement obtained in a series of observations represents the true accommodative power. If, however, this maximum occurs but once, and numerous other observations, made under as good conditions, show a uniformly lower range, we should regard the high finding as probably erroneous.

It is usually the first one or two observations that give the most erroneous findings. This is evidently due to the patient's unfamiliarity with the test and his failure to appreciate just what is expected of him. In any event, it is essential to multiply observations in the same case in order to eliminate error and get at the true result.

If it is thus essential to examine the same patient repeatedly when we wish to determine his individual accommodative power, it is still more essential to multiply observations when we are trying to find the normal average and the normal limits of the accommodation in general. We must examine a great many patients and examine them a great many times and always under the proper conditions and with the proper restrictions. When we do this we find that the accidental variations tend to disappear and the true maxima and minima lie closer to the mean range than we had supposed. The low values, in particular, tend to drop out. It is likely, therefore, that the true limits are not so far apart as indicated in Figure 2 and Table 3, and that a case to be really normal must be well within these limits, and especially must be well above the lower limit.

RANGE IN MYOPIA

Myopia—particularly myopia that has been corrected by glasses—is not regularly associated with a low range of accommodation. Out of thirty cases in which the myopia exceeded 3 D the accommodation was found subnormal in only two. In several cases it was strikingly high; thus:

Dorothy J., aged 18, myopia 12 to 13 D, acc. = 12 D.
B. P., aged 18, myopia 4.25 D, acc. = 14 D.
F. K. M., aged 13, myopia 7 D, acc. = 12 D.
E. C. H., aged 23, myopia 3.75 D, acc. = 14 D.
H. P. R., aged 25, myopia 5.50 D, acc. = 12 D.
A. E. F., aged 37, myopia 5 D, acc. = 8.5 D.

CONCLUSIONS

These may be stated — somewhat dogmatically — as follows:

1. To determine the accommodation we first provide the patient with the glass that fully corrects his refraction. If with this correction his near point lies beyond 40 cm. we add a convex glass of sufficient strength to bring the near point to between 30 and 40 cm. We then place the patient with his back to a window, turning him so that a good diffuse light comes over the shoulder on the side of the eye we are testing — the other eye, of course, being covered. We now put in the carrier of Prince's rule a dull white card 3 by 1.5 mm. in size, mounted on a dead black surface and having engraved on it a vertical black line, 0.2 mm.[5] broad and 3 mm. long. The rod itself we place with its proximal end one-half inch from the cornea and with its broad side vertical. We now push the carrier up to the eye, exhorting the patient to focus on the line as hard as he can. When the line blurs we withdraw it until it is distinct, and then carry it back and forth several times until we are sure we have the very nearest point at which the patient can still see it clearly. Marking this point we now read off from the scale the corresponding amount of accommodation in D. From this we sub-

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[5] If the patient's vision is not sharp, or if his near point is remote, 0.4 mm.

tract the value in D of any convex glass that we may have added to the patient's full correction. The remainder gives the true range. It is necessary to make the test several times, especially in young subjects, getting as many reliable observations as possible. Provided we can be sure that the patient is intelligent and accurate in his observations. the maximum found in a series of tests represents the real range.

2. If the determination is made in this way the range should be between the limits shown in Table 3.

TABLE 3.—NUMERICAL VALUES OF LIMITS FOR EACH AGE.

Normal range in D, near point being measured from the anterior focus of the eye (—1.3 cm. in front of the cornea).

Age.	Mean.	Minimum.	Maximum.	Age.	Mean.	Minimum.	Maximum.
10*	14.0*	11.2	17.5	36	7.0	5.6	8.8
11	13.9*	11.1	17.4	37	6.9	5.5	8.6
12	13.8*	11.0	17.2	38	6.7	5.4	8.4
13	13.6*	10.9	17.0	39	6.5	5.2	8.1
14	13.4*	10.7	16.7	40	6.0	4.8	7.5
15	13.2*	10.5	16.5	41	5.6	4.5	7.0
16	12.9*	10.3	16.1	42	5.2	4.2	6.5
17	12.6*	10.1	15.7	43	4.7	3.8	5.9
18	12.3	9.8	15.4	44	4.1	3.3	5.1
19	12.0	9.6	15.0	45	3.8	3.0	4.7
20	11.7	9.4	14.6	46	3.3	2.6	4.1
21	11.4	9.1	14.2	47	3.0	2.4	3.7
22	11.2	9.0	14.0	48	2.6	2.1	3.2
23	11.0	8.8	13.7	49	2.3	1.8	2.9
24	10.8	8.7	13.5	50	1.9	1.5	2.4
25	10.5	8.4	13.1	51	1.7	1.4	2.1
26	9.9	7.9	12.4	52	1.5	1.2	1.9
27	9.4	7.5	11.7	53	1.4	1.1	1.8
28	9.0	7.2	11.2	54	1.3	0.9	1.6
29	8.6	6.9	10.7	55	1.3	0.9	1.6
30	8.4	6.7	10.5	56	1.3	0.9	1.6
31	8.1	6.5	10.1	57	1.3	0.9	1.6
32	7.9	6.3	9.9	58	1.2	0.9	1.5
33	7.7	6.2	9.6	59	1.2	0.9	1.5
34	7.3	5.8	9.1	60	1.2	0.9	1.5
35	7.2	5.7	8.9	61	1.1	0.8	1.5

* Approximate.

3. An accommodation which is persistently above or persistently below these limits must be regarded as abnormal—provided always that the proper conditions of examination above noted have been fulfilled.

4. We are not, however, justified in pronouncing the accommodation abnormal on the strength of any single observation.

5. The results above given differ from those of Donders in the manner shown in Figure 2 and in Tables 1 and 2, and particularly in the following points: (a) Before the age of 20 the range is lower than that found by Donders; (b) from 20 to 45 it is higher; (c) from 45 on it is lower.

6. We believe that our results are more reliable than those of Donders because of the following facts:

A. They were obtained from three times as many subjects.

B. Our patients were rendered absolutely emmetropic by glasses, and no subject was utilized in whom the total refraction had not been carefully determined; while it is probable that many of Donders' cases, especially between the ages of 20 and 45, had an uncorrected latent hyperopia, which would make his estimate of the accommodation too low by 1 or 2 D. Hence the fact that our curve rises higher than his during this period.

C. The test object that we use for determining the near point (the fine hair-line and the rule) was better calculated to give accurate results than the tape and wire optometer used by Donders.

7. The idea, still somewhat current, that the accommodation in advanced life becomes zero is in accord neither with Donders' findings nor with ours. A true range of 1 D or over exists up to and beyond the age of 60.

8. Myopes have as good a range of accommodation as emmetropes.

49 East Thirtieth Street.

MEMORANDA

MEMORANDA

THE UNRELIABILITY OF THE ASTIGMATIC FAN OR CLOCK DIAL TEST *

DAVID W. STEVENSON, M.D.

RICHMOND, IND.

Nearly all books on refraction give a very large and probably undue amount of space to the astigmatic fan test. There have been a considerable number of later writers who affirm that the astigmatic fan test combined with the fogging method even obviates the necessity for the use of cycloplegics. It is my purpose to prove that the test is very unreliable in a large percentage of cases, and that only by accident is it ever accurate.

It is possible to have the lines appear unlike when the patient has no regular astigmatism. Moreover, it is often true that the lines may look alike in the principal meridians; and yet the patient and victim may still have remaining some astigmatism that ought to have been corrected. Considerable fault may be found with the test, for the additional reason that the average patient, from inexperience and lack of concentration, does not know what is expected of him in the test. Experience has demonstrated these points:

1. If there are three lines, the patient may decide by the middle line, or he may notice only the outside lines. If the lines are partly doubled he may fail to refer to the fact. Blackness is often confused with distinctness.
2. The patient often answers according to a view affecting a peripheral part of the lines and not that covering the macular region.
3. Often adjacent lines are the lightest and darkest; thus 60 may be the lightest and 90 the darkest, while the patient only mentions the most distinct.

*** This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.**

While all will admit that it is easy for the average patient to misunderstand the fan test even if it be founded on correct principles, yet in our further argument we will assume that answers are always given with scientific accuracy. Our contention is that even under conditions so favorable, the fan test is unreliable. As an example, if such a patient with relaxed accommodation requires a correction of plus 2.00 D. sphere, combined with a plus 1.00 D. cylinder, axis 90, it will be true that if a plus 3.25 sphere is put before the eye, both meridians will be myopic; the diffusion image might in such a case resemble a vertical line, and the vertical line on the clock dial will be the most distinct. If a plus 3.00 is used, the assertion will be made that the diffusion image becomes an exact vertical line, but incidentally it may be stated that in the human eye the diffusion image never resembles a geometric figure. With a plus 2.75 the vertical lines will still be the most distinct, while with a plus 2.50 it is stated that it will give an exact circular diffusion circle and thus all the lines on the dial will look equally distinct. Practically such a condition never occurs, as the diffusion image in the best eyes never becomes an exact circle.

With all lenses weaker than plus 2.50 it will be claimed that the diffusion image becomes horizontal and that these lines in the fan become correspondingly most distinct.

One clock dial enthusiast has asserted that in lowering the lenses from plus 3.00 gradually to plus 2.00, the vertical lines, 90 first, become distinct, next the 60 and 120 become the clearest, and later, in order, 30 and 150, and last the horizontal. This is a very gross error because the diffusion image never assumes the shape of a geometric cross with flexible angles merely by a change in spherical lenses. If such erroneous ideas can be believed, no wonder a few optical, correspondence and postgraduate schools may assert that refraction can be learned in a few weeks, and that there is no necessity for cycloplegics.

If the cornea is divided into two halves, either horizontally or vertically, generally it will be found that the center of the diffusion image of each part never corresponds exactly with the center of the other part. In the same way the cornea may be divided into quadrants, or into as many sectors as desired; yet it never can be supposed that the center of the image for each sector lies exactly at the same place as the others.

The serious mistake made by all fogging clock dial enthusiasts lies in supposing that the center of the diffusion image for each sector lies exactly on the theoretic optic axis of the eye. As an illustration, take the case of an eye which can not be improved either by a spherical or cylinder lens, yet in which the upper part of the cornea creates a diffusion image which slightly overlaps that of the lower part (Fig. 1). This overlapping may be compared to that of binary stars which through telescopes are seen to overrlap slightly. It sometimes takes years of observation by astronomers to note if the stars revolve before it can be decided whether or not they are double stars.

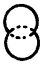


Fig. 1.

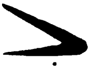


Fig. 2.

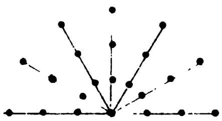
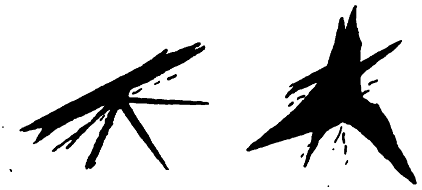


Fig. 3.



Left Eye Right Eye

Fig. 4.

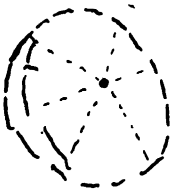


Fig. 5.

With such an overlapping diffusion image vertically placed it may be stated that if the refraction is fogged by placing on plus spheres the vertical lines will yet remain the most distinct. If the attempt is made to force the diffusion image into a rectangular or circular outline by plus cylinders placed vertically, the result will be only disaster because the horizontal part of the image will only be diffused and not rendered more distinct. Minus cylinders would have the same effect if both meridians were fogged. Thus the great evil of the fogging system is in failing to give clear and condensed diffusion images, while merely permitting cylinder-diffused images with a somewhat more circular or symmetrical outline.

Even if millions of eyes were corrected with the best possible spherical and cylinder lenses, the resulting diffusion images would have every possible fantastic shape. No two of these would be alike. Take as an illustration a resulting diffusion image, similar to Figure 2, having an angle with one arm placed at an angle of 30 degrees to the horizontal. In such a case the horizontal clock dial lines and those at 30 always ought to be the most distinct when fogged. If the fan is made up of a series of dots, as in Figure 3, it is easy to see what the result would be by placing Figure 2 over each dot.

Figure 4 shows a drawing made up of the diffusion images of each of my eyes, made while looking at a round electric light seen a mile distant at night time. The vision of each of my eyes is more than double the normal, equal 20/10.

In the study of the refraction of an elderly woman's eyes it was found that she could see fairly well with almost every glass in the trial case. I then asked my assistant to put every sphere in the trial case before her, starting with the strongest plus down, and noting the vision with each. I then had the results platted to scale on quadrille ruled paper. the horizontal lines showing the vision and the vertical lines the strength of the lenses. It was perceived that there were two peaks of best vision several diopters apart, showing a rough test for astigmatism.

I then had the patient make a drawing of the diffusion image of a very small gas jet. It was shaped somewhat like a cartwheel, with a very small rectangular hub, Fig. 5. With the retinoscope and further use of the trial lenses it could now be perceived that there was only a very small central part of the cornea to which attention need be given, and that it acted in her case like a pin-hole disc, and, furthermore, that while the peripheral parts were very astigmatic, they interfered but slightly with the vision. Obviously such a case would merely be muddled with the fan test.

It is impossible to find in the United States or in the world a human eye which does not have irregular astigmatism, while there are many that have no regular astigmatism which can be benefited by a cylinder. Some of the ancients drew their star-shaped figures as having five points, and some six, and others more or less. Irregular astigmatism always tends to render the fan test unreliable and worthless.

The essentials required in careful subjective refraction are a complete trial case including cross cylinders and test letters. The test with these ought to be made in difficult cases with varying illumination and at varying distances. A weak illumination at a short distance may reduce the obscuration by irregular astigmatism, so that the patient may definitely decide what glass gives the most distinct vision. Furthermore, this may require the faithful use of a reliable cycloplegic, combined with further tests made with enlarged pin-hole discs of two, three and four millimeters diameter. If the patient is told to look exactly through the center of these openings, the shadow of these discs will lie concentric with the macula and therefore practically concentric with the pupil.

As the eye is composed of flexible structures, the best results will follow from the study of a wide average, testing the vision with one eye alone and then with both open, alternating with both fogged and later with one at the most distinct focus. It is also well if the tests can be made both before and after the use of a cycloplegic. In many cases these tests require infinite patience and much time and repeated trials, even by an oculist who may have made a prolonged study of every law of theoretical optics.

In visiting my brother, Mark D. Stevenson, at Akron. Ohio, a short time ago, I noted that his office was without a fan test chart. It is true that many well-equipped oculists' offices will be found in which all clock dials, from Green's many varieties to Verhoeff's finer lines, all may have been sensibly consigned to the wastebasket.

In a recent article[1] by D. B. St. John Roosa, some ideas are expressed which might have appeared correct in the middle of the nineteenth century, but which are not consistent with the scientific progress of the twentieth. Following are a few quotations from the article to which exception may well be taken:

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[1] The Proper Method of Determining Errors of Refraction and Their Actual Relation to the Ailments of the Human Body, The Journal A. M. A., Feb. 13, 1909, p. 543.

> 1. I present what I conceive to be safe and sane views on this subject. These views I also believe in the main are those of the majority of ophthalmologists.
>
> 2. The field of abnormal refraction is actually not entered as far as hypermetropia of a low degree is concerned. It is not reached unless it is of a degree higher than, for instance, two diopters, or unless corneal astigmatism is added to it.
>
> 3. I deny the existence of muscular asthenopia, except as dependent on errors of refraction, standing by my views expressed in 1890; I do not find any value in prisms or in decentered glasses, except in cases of paralysis of the ocular muscles, and for years I have not prescribed them, except as above indicated.
>
> 4. To see as well as one can is always desirable, and I have no fear like that so often expressed among the laity, of having glasses too strong either in myopia or hypermetropia, so long as those chosen give the best vision. If they secure this and the patient complains of pain, it may be assumed that the pain is not from the wrong glasses, but from the eye itself.
>
> 5. I do not believe that a difference such as a quarter of a diopter will make any essential difference in the vision of a patient, and especially not in his ability to use his eyes without headache or other asthenopic symptoms.
>
> 6. Unprejudiced judicial examinations made by thoroughly educated and widely experienced men, in my opinion, show the belief that eyestrain causes neurasthenia to be incorrect.
>
> 7. What I believe to be almost infallible evidence against the existence of eyestrain in a given case is the absence of ocular symptoms. . . . In short, before there is eyestrain, there is always some symptoms in the eye itself.
>
> 8. Migraine and neurasthenia will soon cease to be treated through the muscles or refraction of the eye.
>
> 9. "Largely counseled by the late Dr. E. G. Loring, I had abandoned retinoscopy as the most difficult and least satisfactory of the methods of determining the refraction of an eye, which contributes nothing that can not be more easily and more expeditiously performed by the upright image."
>
> 10. Fortunately the use of the ophthalmometer is easily learned. With a good teacher, in two weeks' daily practice, a man at all skilled by education and experience in other manipulations and observations will be able to go by himself.

As to the second quotation, it may be stated that of all glasses ever given, probably more benefit has resulted from a hyperopia corrected of less than 2.00 D. than of any spherical type. In numberless cases patients of this class have truthfully told their oculists that if their glasses could not be replaced, no money could buy their present ones, as life would be considered unendurable without them.

As to the third statement, it is within the bounds of absolute truth to say that in such conditions as hyperphoria and allied conditions prisms have been of untold value.

Neurasthenia may have many causes, from fatigue to auto-intoxication, but eyestrain is undoubtedly one of its most prolific causes in this age of overworked vision. As to the severe migraines such as have a distinct heredity and compel the patient to go to bed, a considerable proportion are not relieved by glasses; yet there remains a large percentage of migraines which have been entirely cured by careful refractive work.

As to the comparison between the objective methods of testing, such as the ophthalmometer, the retinoscope and the direct method, it can be truthfully said that the retinoscope is incomparably better than either of the others, as its measurements can be taken from the macular region.

The axis of astigmatism is seldom definitely shown with the direct method and is always uncertain. The ophthalmometer shows only the anterior part of the cornea and therefore it is totally incomplete because it fails to give the lenticular astigmatism, while that of the aqueous, vitreous and posterior layer of the cornea are neglected. The statement that the ophthalmometer takes the place of cycloplegics is not worthy of the least consideration and ought not to pass unchallenged.

Almost every ophthalmologist of fifteen years' active study and practice will affirm that during the last years of his practice his refractive results have improved marvelously, while his former tests were something to be surprised at and possibly ashamed of. In no branch of his study and life-work has more real gain been made by experience and constant education than in his personal refractive training. Refraction is the work of a lifetime and a study that is never completed.

MEMORANDA

SIMPLE FIBROMA OF THE ORBIT *

WILLIAM E. GAMBLE, B.S., M.D.
Professor of Clinical Ophthalmology and Associate Professor of Ophthalmology University of Illinois.
CHICAGO

Simple fibroma of the orbit I take to be one that springs from the retrobulbar connective tissue, i. e., the orbital fascia within that cavity. Orbital fibroma commonly arises from the periosteum of the orbital walls and less commonly from the sheath of the optic nerve; occasionally from the capsule of the lachrymal gland and rarely from the tendons of the muscles (G. Mizno[1]).

Marchetti[2] reports a case of "peripheral fibroma" which developed on the infraorbital nerve "causing elephantiasis of every part of the skin which was supplied by the compressed nerve." It is altogether probable that this tumor should have been classified as plexiform neuroma.

A few reports have been made of subcutaneous fibroma of the eyelids (Mizno[1]).

Fibromata of the skin of the eyelids occur in general fibromata (Oliver[3]).

Heretofore it has not been definitely determined that fibroma is ever derived from retrobulbar connective tissue. Lagrange has collected reports of cases which, to more or less extent, argue such origin.

In my opinion, Goldzeicher[4] comes the nearest to proving the point. He reports a case of freely movable

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[1] Mizno, G.: Nippon Gankwa Gakukwai Zasshi, 1904, v, No. 8.

[2] Marchetti: Ann. di ottal. e Lavori d. Clin. ocul., Naples, 1904, xxxiii, 29.

[3] Oliver: Ophth. Rec., viii, No. 6.

[4] Goldzeicher: Centralbl. f. prakt. Augenh., 1894, xviii, 65.

encapsulated tumor in the orbit in a male, 17 years of age. A report of the microscopic examination is as follows:

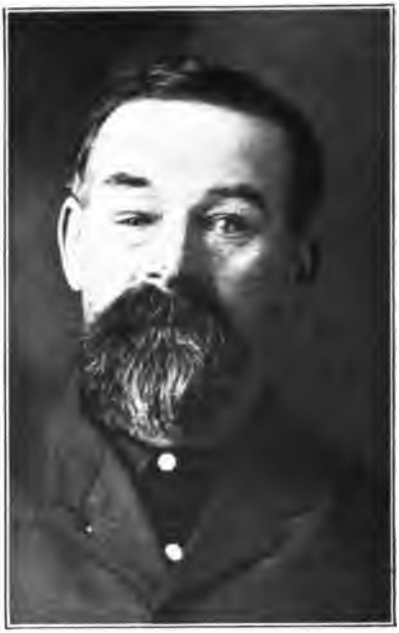

Fig. 1.—Photograph taken 1 year before operation. Deformity not so great as at time of operation.

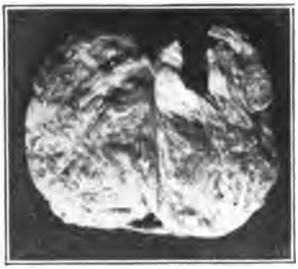

Fig. 2.—Tumor split in halves, showing cut surfaces, hinging on capsule. Gaping portion indicates part removed for section.

Hard connective tissue fibers bound the tumor together, containing sparse, spindle-shaped cells. Fibers generally run parallel; in spite of the hardness of the tumor contain many

blood vessels of all shapes, with endothelium; fibers of the periphery of the capsule over the posterior orbital surface were compressed and poor in cells. The impression given is that the neoplasm arose from a vein.

A most careful study of the microscopic section is necessary in order to differentiate between endothelioma and fibroma. It is in the hope of shedding light on this mooted question that the following case is here reported:

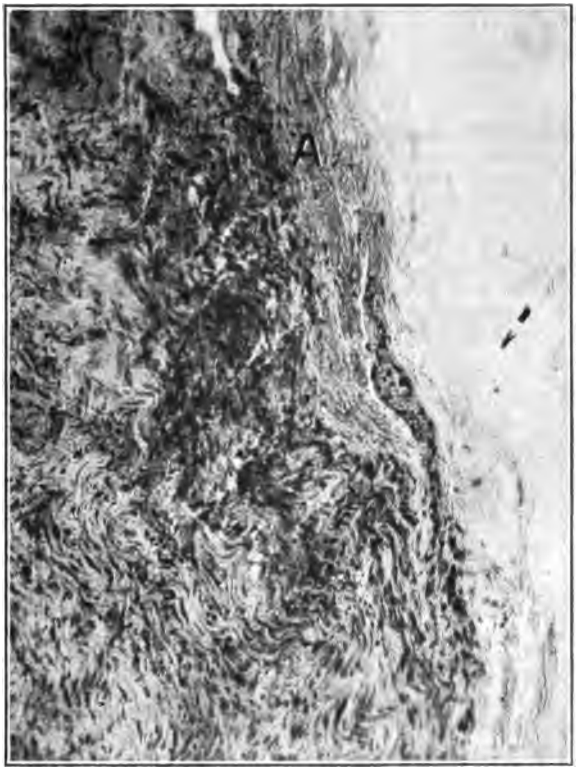

Fig. 3.—Low power: X 125; *a* indicates capsule.

Patient.—**Mr. L., aged 55 (Fig. 1), came under my observation Aug. 21, 1905, on account of a drooping of the right eyelid, he having been referred to me by Dr. F. C. Dielman, Fulton, Ind. The patient first noticed this deformity three years before. During the previous six months the**

deformity had rapidly become worse. There was no history of injury or other disease of the eye. The eye was displaced downward and forward. There was a dull ache when the patient was out in the wind. Vision was 20/20. There was no change in the fundus. Deep pressure with the little finger between middle of the upper orbital margin and the eyeball revealed a hard movable mass which was not tender on pressure. There being no history or evidence of syphilis, I advised an operation.

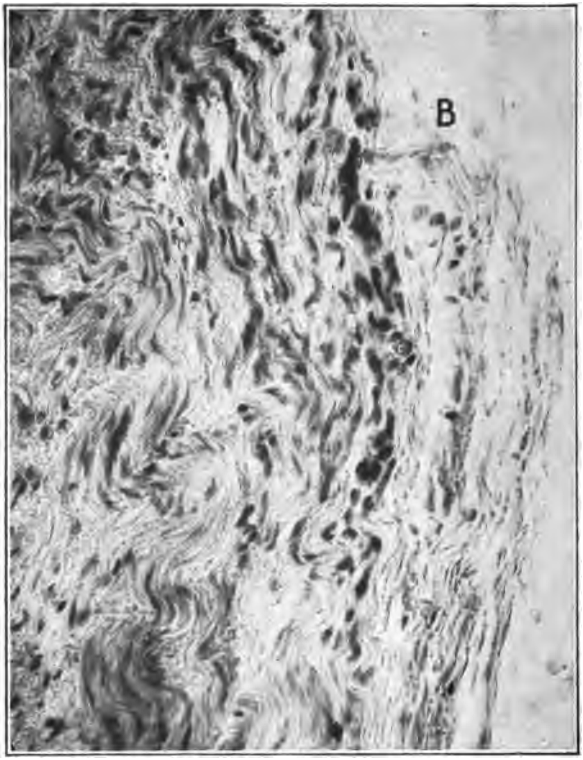

Fig. 4.—High power microscopic view of same section. X 300; *b* indicates capsule.

Operation.—On August 22 an incision was made in the brow, conforming to the supraorbital margin, down to the periosteum. The orbital fat being dilacerated by a grooved director and the pointed scissors, a white, hard, encapsulated tumor about the size of a small hickory-nut came into view (Fig. 2), which was pulled forward and with the aid of the director and fingers was readily enucleated, there being no connection with the periosteum, optic nerve, or tendons.

Tumor.—Microscopic examination (Fig. 3) showed it to be a pure fibroma, homogeneous throughout. Few blood vessels were present. Parsons[5] says that the best test of simple fibroma would seem to be encapsulation, fibrous tissue composing the growth being matted together at the periphery. How well this tumor meets this test is shown by the accompanying photomicrographs (Figs. 3 and 4).

Postoperative History.—Nearly four years have now elapsed since the operation. No recurrence has taken place. The eye is restored to its normal position and the cosmetic effect is all that could be desired.

I am under obligations to Dr. L. W. Grosvnor, Dr. W. A. Walters for section of tumors, and to Dr. C. G. Darling for photograph.

103 State Street.

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[5] Parsons: Pathology of the Eye, II, 228.

MEMORANDA

DROPSY OF THE OPTIC NERVE SHEATH *

JOHN A. TENNEY, M.D.

BOSTON

Much has been written concerning optic neuritis since von Graefe supposed it to be caused by obstruction in the cavernous sinus, caused by intracranial pressure. The discovery of anastomoses between the ophthalmic and facial veins made that theory untenable. Schmidt-Rimpler and Manz held that the outflow of the venous blood was impeded by pressure on the optic nerve caused by dropsy in its sheath, and as a consequence stagnation of lymph in the papilla. Leber and Deutschmann believe that the neuritis is caused not so much by the pressure as by the toxic quality of the cerebrospinal fluid, which is loaded with the products of tissue change. These theories depend on pressure as the prime factor.

On the other hand, Gowers,[1] Brailey,[2] Edmunds,[3] Mackenzie[4] and others believe that the disease is not due to pressure at all, but to a descending inflammation transmitted from the intracranial cavity. Those who support the mechanical-pressure hypothesis think that it is an ascending process, while those who hold the opposite view think that it is a descending neuritis.

Leber[5] records a case of a patient hydrocephalic from birth, liable to frequent and severe headaches, occasional epileptic attacks and marked loss of sight. At twenty an almost continual dropping of water from the nostril came on, and with it came great relief from the vertigo and epilepsy. The liquid was found to be identical

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[1] Diseases of the Brain.

[2] Tr. Internat. Med. Cong., 1881, iii, 63.

[3] Tr. Ophth. Soc. U. Kingdom, i, 112.

[4] Brain, ii, 257.

[5] Arch. f. Ophth., xxix, 273.

with the cerebrospinal fluid. Similar cases had been reported before by Nettleship,[6] Priestley Smith, Elliotson,[7] Baxter,[8] Paget[9] and Jones,[10] the patient of the latter observer being sixty-seven years old. In all these cases the head symptoms were aggravated when the dropping ceased. Autopsies were held in the cases of Paget and Jones, but no intracranial disease was found.

Leber thought that the cerebrospinal fluid must have passed through a minute opening in the ethmoid bone; but, as there is a normal passage between the third ventricle and the subarachnoid space, it is more probable that the fluid followed the sheath of the olfactory nerve.

Schwalbe many years ago found a communication between the subarachnoid space and the intravaginal sheath of the optic nerve, and it has been demonstrated in recent years that liquids can be forced through these passages artificially. Levinsohn[11] injected cinnabar into the subarachnoid space in rabbits and cats, and later found this substance in the intravaginal spaces of the nerve and along its blood vessels. Kruedener[12] demonstrated in five cases that optic neuritis disappeared after the trephining process, and Abadie[13] reports a patient cured by lumbar puncture.

Case 1.—Six years ago a young woman with light hair and fair skin came to me with double optic neuritis. The vision in the right eye was 3/60, while the other eye could only tell where the windows were in the room. She said she had been troubled with this loss of sight for a year. She had consulted noted specialists in New York and Boston, all of whom told her she would never see again. She vomited every day, often fell down while walking on the street, and had terrible headaches. The length of time that had elapsed since the beginning of the trouble seemed to exclude cerebral tumor, or it must have been of slow growth, it seemed to me. Instead of getting discouraged over the case as others had done, I resolved to act on the assumption that possibly intracranial pressure had produced this state of things. Accordingly, I prescribed potassium iodid in saturated solution, beginning with ten drops three times a day, and increasing by five drops each day. When the patient was taking fifty drops three times a day the characteristic eruption covered her body. The dose was reduced to twenty drops, and arsenic was given to relieve the eruption. In six months she could see 20/20 with either eye.

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[6] Ophth. Rev., January, 1883.
[7] Med. Times and Gaz., Sept. 19, 1857.
[8] Brain. January, 1882.
[9] Tr. Clin. Soc. London, 1878, xii, 43.
[10] Ophth. Rev., vii, 97.
[11] Arch. f. Ophth. (Graefe's), lxiv, 3.
[12] Arch. f. Ophth. (Graefe's), lxv, 1.
[13] Clin. Ophth., April 10, 1903.

The treatment in this case was suggested by my experience with a number of patients who came with loss of vision, on whose cases no history would throw any light and no ophthalmoscopic appearances came to my aid. It is in the interest of such patients that this paper is written; for, although medical literature is full of details concerning optic neuritis, I do not find such cases mentioned.

Case 2.—The first of my patients was a schoolgirl of 15, sent by a physician in Plymouth county, fifteen years ago. She said that she had lost the vision of the right eye, and was positive that she had not accidentally discovered blindness that she had had from birth. The ophthalmoscope revealed nothing. The retinal veins looked as well as they do in most cases. On the assumption that pressure on the optic nerve might be the cause of the trouble, potassium iodid was prescribed in moderate doses. In a week the patient could see as well as ever.

Case 3.—In a year or so a girl of the same age came from Maine with the same trouble in the right eye. She said she became blind in that eye the first of May. As it was then the middle of June, there seemed to be little improvement. She was given the iodid, and in ten days she went home. A year afterward she came with the vision more seriously impaired than it had been before. When she looked at a lamp in a dark room she saw a light mist. She said that she had taken the medicine she had before, without effect. The dose was increased, and in ten days she went home, and has not been heard from since; the inference is that she has had no further trouble.

In neither of these cases was there any evidence of menstrual or other constitutional disturbance.

Since that time I have seen eight similar cases. I have never used pilocarpin, thinking that the iodid might, with its alterative action, better prevent the return of the disease. In all these cases, marked with sudden loss of vision, with or without headache, in which the ophthalmoscope revealed no changes in the retina, and in which no history threw any light on the trouble, perfect recovery took place.

2 Commonwealth Avenue.

MEMORANDA

REFLEX AURAL NEUROSES CAUSED BY EYESTRAIN

WITH REPORT OF CASES *

SAMUEL THEOBALD, M.D.
Clinical Professor of Ophthalmology and Otology, Johns Hopkins University; Ophthalmic and Aural Surgeon to the Johns Hopkins Hospital and to the Baltimore Eye, Ear and Throat Charity Hospital
BALTIMORE

Although nearly all the ills that flesh is heir to, not excepting dysmenorrhea, dysuria, sterility and habitual constipation, have been ascribed to eyestrain, but scant consideration has been given to the influences which, not very rarely, it exerts on the auditory apparatus. So scant, indeed, is this consideration that, though I am sure the matter has been treated of, I have been unable to find mention of it in any treatise on diseases of the eye or the ear at my command, or a single reference to it in the titles of papers contained in the Index-Catalogue of the Surgeon-General's Library.

I have heretofore, in enumerating the less common consequences of eyestrain, mentioned tinnitus aurium as one of them,[1] and in the discussion of a paper on "Objective and Subjective Tinnitus," presented to the American Otological Society, in 1896, by Dr. Alexander B. Randall, I reported a case of this character observed several years previously.[2] Since this first observation the subject has been of interest to me, and I am convinced from the number of cases of similar character which I have encountered in the interim that it is one deserving of greater consideration than it has hitherto received.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

[1] Prevalent Diseases of the Eye, p. 390.

[2] Tr. Am. Otol. Soc., xi., 365.

While tinnitus is the aural reflex that I have most frequently met, as a result of eyestrain, it is not the only one. Others that may be mentioned are a "muffled" or "stuffed" sensation in the ear, pain, not severe, and as often felt around and in front of as in the ear, and impairment of hearing, which the tuning fork seemed to indicate was due to disturbance of the perceptive rather than of the conducting apparatus. Whether, as well, the vertigo recognized as a by no means unusual symptom of eyestrain, and which was present in several of my cases, deserves to be regarded as an aural reflex I am not prepared to say, though it seems not at all improbable that the derangement that gives rise to it is in the semicircular canals.

Of tinnitus due to eyestrain I have observed three distinct varieties—the more usual or vascular type, the relatively low-pitched, whirring or fluttering sound caused by irregular contractions of the tensor tympani, and the high-pitched, almost musical, intermittent tinkling produced by like contractions of the stapedius muscle.

The evidence in favor of the ocular origin of the aural sensations enumerated is, first, their disappearance after relief of the eyestrain; second, that the ear affected—for the sensations were commonly unilateral—was usually the one on the side of the more troublesome eye, and, third, that they often became more pronounced when the eyestrain was most annoying, or were brought on, or at least intensified, by use of the eyes. The ocular fault often present was astigmatism, sometimes of high degree and associated with other marked refractive errors, though in some instances it was chiefly heterophoric.

Politzer remarks, in his exhaustive and interesting discussion on "Subjective Sensations of Hearing," that such sensations are "occasionally produced reflexly from the trigeminus,"[3] and this doubtless is the explanation of those which are caused by eyestrain.

The nerve supply of the tensor tympani is derived from the motor branch of the fifth nerve; the stapedius receives a twig from the seventh, and it is as easy to comprehend how eyestrain may produce irregular con-

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3. Diseases of the Ear. Lea Bros. & Co., p. 772.

tractions in these two muscles as in the orbicularis, which, as is well known, it often does.

The more subjective and more usual varieties of tinnitus, when caused by eyestrain, as well as the impairment of hearing which sometimes coexists, are to be explained, in all probability, by a vasomotor disturbance of the intralabyrinthine blood vessels. The "muffled" or "stuffed" sensation in the ear, not infrequently complained of, may be similarly explained, or, like the otalgia, may be due simply to a reflex irritation of aural branches of the trigeminus, of which familiar examples, such as otalgia dentalis, might be cited.

I have the notes accessible of about twenty cases of subjective aural sensations which, it seemed probable, were produced, in part at least, by eyestrain. I shall, however, describe briefly but four of the most striking of these, and first I may speak of a personal experience.

CASE 1.—Having inherited what may be termed a very intractable pair of myopic and astigmatic eyes, I have had occasion to adjust and readjust my glasses oftener than I care to confess. About sixteen years ago I developed a very subjective, one-sided tinnitus, which persisted for some weeks despite my efforts to rid myself of it. Finally it occured to me that my eyes, which were giving me trouble at the time, might be responsible for it. I, therefore, readjusted my glasses, and the tinnitus promptly disappeared. Some months afterward it returned, and disappeared as quickly as before with another change of glasses.

Several years elapsed without further aural sensations, when a tinnitus of a different character made its appearance—an intermittent, whirring or fluttering sound, less subjective than the tinnitus, previously experienced—which I made no doubt was caused by irregular contractions of the tensor tympani. This, too, promptly ceased when my glasses were once more modified.

More recently, with a return of asthenopic symptoms, I became aware of an intermittent clicking or tinkling sound, of high pitch, in the right ear, the previous sounds, if I remember rightly, having been heard always in the left ear. Sometimes there would be a single click, sometimes two or three in rapid succession, and again a longer succession of clicks—a not unpleasant, semimusical metallic tinkling. The sensation was much less annoying and entirely unlike that previously experienced from irregular contractions of the tensor tympani, and was, I feel sure, caused by like contractions of the stapedius muscle. It may be of interest to add that I have in times past been annoyed by spasmodic twitching of the palpebral fibers of the orbicularis palpebrarum.

CASE 2.—Mr. N., aged 19, a student in Johns Hopkins Medical School, complained of asthenopia, chiefly in right eye, a "stuffed" feeling in right ear, a numb sensation around and in front of this ear, and occasional giddiness. Choreic contractions of the right orbicularis palpebrarum, of the levator of the upper lip on the same side, and of some one of the small muscles having to do with the nose, which caused perceptible movements of his eyeglasses, were subsequently observed.

Examination.—He was found to have a low grade of hypermetropia, slightly greater in the right eye, and a very high degree of left hyperphoria.

Treatment.—Notwithstanding the great amount of hyperphoria shown by the tests—from 10 to 16 degrees in distant, and from 20 to 22 degrees in near vision—he was not inclined to accept a very considerable prismatic correction. Spheroprismatic lenses correcting his manifest hypermetropia and but 4° of the hyperphoria were, therefore, prescribed. These for a time afforded a measure of relief. Subsequently, I performed a "guarded" tenotomy on the left superior rectus, reducing the hyperphoria by about 8°, and this, supplemented by the glasses, which he continued to wear, was followed by more decided relief as to both the asthenopia and the reflex symptoms.

Result.—The relief, however, was not complete, and has not proved lasting, and as the residual hyperphoria is still marked, I am waiting for an opportunity of a short rest from work, which he will soon have, to tenotomize the right inferior rectus. I may add that the patient notices a definite improvement in the aural manifestations from even a brief rest of the eyes from near work, and, further, that his hearing is not impaired.

CASE 3.—Mrs. R., aged 37, consulted me in November, 1908, complaining of asthenopia, headache, giddiness, a "stuffed" feeling in the ears, and of bilateral earache, which had manifested itself only a short time previously. She was found to have compound hypermetropic astigmatism, and glasses, for constant use, were prescribed as follows:

R. eye + .62s $\overset{\frown}{\smile}$ + .25c 100°
L. eye + .50s $\overset{\frown}{\smile}$ + .37c 100°

She was requested to report the effect produced, and, on January 5, wrote me: "I have found a great deal of relief from headache and dizziness by the constant wearing of the glasses. I have suffered no inconvenience from earache, and have at no time since seeing you noticed any muffled feeling in my head."

CASE 4.—Mr. S., aged 45, of distinctly neurasthenic temperament, complained of tinnitus, occasional giddiness, a "stuffed" feeling in the ears, nervousness, and "severe pain in the head." Attempts to use the eyes in reading, he said, increased the tinnitus and nervousness. The pain was more severe on the right side of the head, the noise louder in the right ear, and in addition to the tinnitus, which was constant, he occasionally experienced a "clicking" sensation, usually in this same ear.

Examination.—He was wearing, in far and near, + 1.s lenses, prescribed by a well known New York oculist. His hearing was practically normal for watch and voice, and the tuning fork showed nothing worthy of note. With his glasses on, the muscle balance was normal, except that he had only 1/2° of exophoria at the reading distance. Compound hypermetropic astigmatism, against the rule, was easily made out; but, as the ciliary muscles were very irritable, a cycloplegic was employed.

Treatment.—As a result of several tests, glasses were prescribed for distance as follows:

R. eye + 1.75s $\overset{\frown}{\smile}$ — .50c 95°
L. eye + 1.75s $\overset{\frown}{\smile}$ — .37c 75°

and for reading the sphericals were increased in strength .75D. Three months later the correction for distance was changed to:

R. eye + 2.37s $\overset{\frown}{\smile}$ — 1.12c 97½°
L. eye + 2.12s $\overset{\frown}{\smile}$ — .50c 70°

and + .62s was added for near vision.

Result.—As a result of this change, his asthenopia and head pains were relieved, and after a few weeks, ammonium chlorid having been administered in the meantime, he reported the tinnitus as appreciably lessened, and as having become intermittent.

Subsequent History.—Nearly four years elapsed without the patient having further occasion to see me, when a return of asthenopia from near use of the eyes, occasional headaches, failing vision for small print, and "some tinnitus," which he was not disposed to admit he had ever been entirely rid of, led him to seek further advice. An increase of .12 D in the astigmatic correction of the right eye was found to be indicated and, of course, a considerable strengthening of his presbyopic lenses. This was two years ago and he has not consulted me since. In reply to a recent letter addressed to him, he writes that the "noises and stuffy feelings" are better, but that he is not entirely free from them. He adds that he is now under the care of a well known psychiatrist, whose treatment has given him a great deal of relief.

ADDENDUM

When the writing of this paper, except the notes of cases reported, had been completed, Dr. H. O. Reik, whom I had asked to assist me in searching the literature for anything that might have been published having reference to the production of subjective aural sensations by eyestrain, brought to my attention the paper of Dr. F. Park Lewis,[3] of Buffalo, New York, on "The Ophthalmic Origin of Otic Disturbances, with the Cerebellar Control of the Eyes." I was much interested to find my observations confirmed by those of Dr. Lewis and to note the close similarity between some of his cases and some of my own. And especially was I interested to find mention in his enumeration of aural symptoms of the sensation of "stuffiness" in the ears which I have so frequently encountered, and which, I think it will be admitted, is not a symptom often complained of in purely aural affections.

Dr. Lewis' search of the literature bearing on "The Ophthalmic Origin of Otic Disturbances," as he phrases it, evidently has been more thorough and more successful than my own, for he mentions a case of this nature described by Dr. George T. Stevens in a paper read before the International Medical Congress in 1881; a paper by Dr. Arthur A. Boyer on "The Relative Importance of Labyrinthine and Ocular Defects in the Etiology of Vertigo," presented to the Neurological Section of the New York Academy of Medicine in 1895, in which the author cites ten cases of "vertigo and lowered hearing" associated with refractive and muscular faults, and which were relieved by their correction, and, finally, a more recent article by Dr. Marcel Rollet,[4] of Blois, "De l'influence de l'oeil sur l'oreille *à* l'état normal et pathologique."

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[3] Ann. Ophth., January, 1909.

[4] Rev. hebd. December, 1908.

MEMORANDA

MEMORANDA

AMETHYST-TINTED LENSES

A PRELIMINARY COMMUNICATION*

L. WEBSTER FOX, M.D., LL.D.
PHILADELPHIA

HISTORICAL DATA

While man has always instinctively protected his eyes from the harmful influences of light, no reliable historical data are available to demonstrate the evolution of the use of colored glasses. Like many other customs originally based on empiricism, the subject is inextricably blended with the mists of antiquity. Ever since ophthalmology has become an integral part of medical science, ophthalmic surgeons have been confronted with the problem as to what color is best incorporated with glasses for the purpose of protecting the eyes against the various harmful influences attributed to light, quantitatively as well as qualitatively. Up to a comparatively recent time the injurious effects were supposed to be due to excessive luminosity, but we know that the ultraviolet rays are chiefly responsible. The problem is not only of importance from a scientific standpoint, but is assuming economic proportions of no small degree, when we consider the number of cases and the virulence of the symptoms that go hand in hand with the increasing employment of artificial sources of illumination.

A century ago green glasses were worn. Later these were superseded by blue glasses of the same tint as the sky. As these were easy to manufacture in all dark gradations of color and comparatively inexpensive, they became popular with the laity, but otherwise acquired very little status. Glasses of neutral tint or smoke-color have afforded considerable relief, although the glasses were sometimes made so dark in color as to impede decidedly the power of vision.

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In 1887 Fieuzal,[1] after numerous investigations, introduced a combination of gray, yellow and green, which still bear his name under the term "Fieuzal glass." This glass was so colored, as the result of the investigations of Van Genderen Stort,[2] who showed experimentally that the pigment, as well as the movement of the retinal elements, were reduced to a minimum under the influence of yellow light. The glass was not intended to arrest the ultraviolet rays, as their harmful effect upon the eye was not yet known at that time. While these glasses have been found acceptable and soothing, they afford no protection against artificial sources of illumination, which are rich in ultraviolet rays, as for instance, the electric arc lamp. Various other tints have been introduced from time to time, many of which, although withstanding spectroscopic and other tests, have failed on practical application.

In 1887 I was first led to use the amethyst-tinted glasses on noting that dentists, by constant use of their eyes in filling teeth with gold, became gold blind, failing to differentiate the walls of the tooth and the gold, and could continue their work only after a certain period of rest. It occurred to me that the yellow of the gold was producing over-stimulation of the retina and that a possible remedy would be the amethyst-tinted glass found in some of the old Philadelphia windows. Some of this glass was procured and I had a pair of spectacles made for a dentist patient which promptly relieved him and enabled him to pursue his work continuously for days without intermission. This good effect was confirmed in many other cases, and I next began to investigate the phenomenon of the coloration.

THE COLORATION OF GLASS FROM CONTINUED EXPOSURE TO LIGHT

After some window glasses have been exposed to intense sunlight for some time a violet coloration frequently develops, varying from a faint to a deeper color according to the time of exposure. The images of letters which have been removed from show windows often appear colorless against a background having a violet coloration. In 1905 Sir William Crookes presented to the Royal Society two panes of glass which had been colored a deep violet as a result of having been exposed to sunlight in South America at an altitude of almost 1,500 feet. On account of the increasing intensity of sunlight, the higher the altitude, it seems probable that the ultraviolet rays were responsible for the coloration. Indeed, it has been shown that glass can be colored violet after having been exposed for forty-eight hours to the ultraviolet rays emanating from the radiation of a mercury vapor lamp.

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[1] Bull. de la clinique nationale ophthal. de l'hospice des Quinze Vingts. 1887. v. No. 2; 1885, iii, No. 3.

[2] Arch. f. Ophth. (Graefe's), 1887. xxxiii, 3.

At one time it was supposed that the coloration was due to traces of manganese which glass manufacturers add to the glass to overcome the yellowish tint derived from the presence of iron, but this theory has been practically exploded. According to Ladenburg the coloration is the result of the formation of manganic acid from the ozone which the rays originate. But this theory is refuted by the fact that we also find the coloration inside of a Crookes tube in which the amount of oxygen is too small to be taken into consideration. It is now held that the coloration produced by ultraviolet rays, radium, *x*-rays, cathode rays, and the Becquerel rays from radioactive substances is due to very minute (ultramicroscopic) particles of metallic potassium or sodium in the glass.

DISTRIBUTION OF THE GLASS IN THE UNITED STATES

It may be interesting to note that I have found specimens of glass which had been changed from a white to an amethyst color by the sun in different parts of the country. Some I found in Philadelphia windows, and it was from these specimens that the evolution of the amethyst-tinted lenses took its inception. These window panes were brought from England seventy-five years ago, and at that time were white and clear, like any ordinary glass; but in due course of time their color has changed to a beautiful amethyst tint, and the singular fact has manifested itself that these changes only took place in the windows on the north side of streets running east and west, or else on the corner of the street where the glass was exposed to the sun. I have found other specimens of glass in Montana, Colorado, Florida, Maine, New Hampshire, New Jersey, and other states. While in Estes Park, Colorado, last summer, through the courtesy of Mr. Enos Mills, I learned that it required eighteen months to change an ordinary glass cup (white) to an amethyst tint No. 1. Mr. Mills purchased this glass cup and placed it near a spring on Long's Peak where it was constantly exposed to the sun's rays for the time mentioned, at an altitude of 12,000 feet.

THE EFFECT ON THE EYE OF ULTRAVIOLET RAYS FROM ARTIFICIAL ILLUMINATION

Artificial illumination, especially when considered from an economic standpoint, is undoubtedly the chief factor responsible for the various ocular affections attributed to the ultraviolet rays. In all large industrial establishments requiring the employment of artificial light, in all forms of passenger transportation in which electricity is used for illuminating purposes, in modern dwellings, and in the illumination of thoroughfares of all large cities as well, it has been the object to increase the luminosity of electric light appliances. The result has been that the apparent disregard of the effects on the eyes has caused an increasing number of cases, varying from simple conjunctivitis to profound ocular disturbances, as the result of electric ophthalmia. Before the introduction of electricity for illuminating purposes the cases were largely confined to accidental exposure to direct sunlight, as the viewing of solar eclipses with unprotected eyes, snow blindness, or from lightning, etc. We now find the cases increasing both in number and virulence among clerks, typewriters, compositors, printers, seamstresses, and, in fact, in all vocations requiring close application under artificial light.

While the ultraviolet rays generally cause intense conjunctival irritation, the crystalline lens, as a rule, prevents them from affecting the retina, the lens itself becoming intensely fluorescent, as originally shown by Widmark. That this fluorescence is to be attributed exclusively to the ultraviolet rays is proved by the fact that if a glass which will absorb the rays, such as the euphos glass discovered by Schanz and Stockhausen,[3] is placed between the eye and the source of radiation, the fluorescence disappears, only to reappear again when the glass is removed, and at the same time there occurs a reflex closure of the lids in the animal. It is now recognized that the spectrum is lengthened toward the ultraviolet side after cataract extraction, and that the erythropsia occuring in these cases is due to the influence of the ultraviolet rays on the retina. It may also occur, however, in normal eyes, as Fuchs and others have observed, when a dark hut is suddenly entered after prolonged trips among glaciers. Experiments made on the aphakic eyes of rabbits by Birch-Hirschfeld[4] have shown that, after the eyes have been exposed from half to three-quarters of an hour to the ultraviolet rays from an electric light, retinal changes appeared, characterized by a solution of the chromatin substance of the ganglion cells, the formation of vacuoles in the protoplasm, and loss of chromatin in the nuclei of the inner nuclear layer, and a bleaching and change in structure in the nuclei of the outer nuclear layer. The absorption of ultraviolet rays by the lens has furthermore been substantiated by Schulek, Hertel and others.

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[3] Arch. f. Ophth. (Graefe's), lxix, 49.

PROTECTION OF THE EYES FROM THE ULTRAVIOLET RAYS

The large number of theoretical data which have been obtained in the search for the proper coloration of glass as a means of protecting the eye against ultraviolet rays, particularly those of from 300 to 400 mm. wave length, largely depend on animal experimentation, the human eye being obviously unfit for such investigations. And yet, after all the painstaking experimentation on the part of such singularly astute observers as F. Schanz and K. Stockhausen,[3] the astonishing assertion is made by them that, although their euphos glass succeeds in absorbing the ultraviolet rays, it is more practical to use it for the manufacture of lamp chimneys, electric light bulbs, etc., than to employ it for the manufacture of spectacle lenses. Furthermore, while the discovery of this glass fulfills the purpose of protecting the eyes from ultraviolet rays, it is admitted that it is not a matter in indifference if the eye is entirely removed from the influence of the rays.

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[4] Trans. German Ophth. Society, Heidelberg, Sept. 14, 1903.

While theoretically, and scientifically, much has been done toward solving the matter, the practical side of the question is, after all, the one toward which we must look for an answer, once more illustrating, what has so often been found in the development of medical science, that an empirical fact is often the forerunner of a scientific discovery. Vaccination was discovered long before the theories of immunity were established on a scientific basis, nor could it wait for a theoretical corroboration; quinin was discovered as a specific for malaria long before the microscope became trained on its etiologic factor; likewise many therapeutic measures were discovered and successfully employed long before their scientific status had been established.

AMETHYST-TINTED LENSES

As stated above. the amethyst-tinted lenses had their inception in my observations of the sun-colored glass. The only mention in literature that I have been able to find relating even to the experimental use of window glass is that of Widmark, who found that the interposition of a piece of window glass 2.5 mm. in thickness between the source of light and the eye lessened the severity of the symptoms. It does not state, however, whether this piece of window glass had previously been exposed to action of sunlight. F. Schanz and K. Stockhausen also tried the experiment with a piece of plate glass 18 mm. in thickness, using an arc lamp very profuse in its ultraviolet radiations, but in their experiments the symptoms were not materially influenced. It is possible that the plate glass in their case had never been exposed to sunlight, while the window glass of Widmark probably had.

As the amethyst window glass of Philadelphia is becoming continually scarcer, efforts are now being made to procure the coloration by artificial means. I am not yet prepared to state whether the artificial glass will have the same therapeutic effect as that naturally colored, but this will be a subject of future observation.

I have graded the color into various shades, calling them 1, 2 and 3. respectively, and am using them according to the manifest clinical conditions.

The glass can be used in the manufacture of lenses of all foci and curvatures.

THERAPEUTIC INDICATIONS AND RESULTS

The results which I have obtained from amethyst-tinted lenses date back to their employment in the case of dentists afflicted with goldblindness and cover an experience of over twenty years. In cases of asthenopia the results have been most gratifying. In the case of printers, typewriters, students, seamstresses, and all those who are required to apply the eyes closely under artificial illumination, prompt and permanent relief was afforded, especially when the amethyst tint was incorporated with correcting lenses. Especially in asthenopia of anemic women the results were most satisfactory; like results were obtained in other cases of ocular fatigue. No. 1 tint is prescribed in all mild forms of asthenopia; No. 2 and 3 in severer forms. As the severe cases become relieved I change to No. 1 color, and these are then worn indefinitely. After wearing even the pale tint the patient does not care to go back to the colorless glass. The glass has been prescribed with great relief to those whose eyes are exposed in *x*-ray laboratories. In one case a patient of mine who had been almost blind for red, as a result of continually working on that color in a factory, was promptly and permanently relieved by amethyst tint No. 3. On my recommendation many of my friends have prescribed this glass and have met with the same beneficial results.

I have requested various opticians of Philadelphia to furnish lists of the number of prescriptions for amethyst lenses. filled during the last five years, and as nearly as we could ascertain there were about three thousand. A majority of these prescriptions were given by Philadelphia ophthalmic surgeons and in many instances they came from elsewhere. The records also show that the demand for these glasses is increasing monthly.

Whether the production of the amethyst tint by sunlight or its reproduction by artificial means imparts a quality to the glass which acts as an immunizing agent against the harmful effects of excessive ultraviolet rays, or whether the beneficial results obtained depend on purely spectroscopic principles. future observations will determine.

1304 Walnut Street.

MEMORANDA

SOME MINOR POINTS IN THE SURGERY OF CATARACT *

HOWARD F. HANSELL, M.D.
PHILADELPHIA

Methods for the successful extraction of cataract have been so often the subject of discussion that it would seem as though further discussion were superfluous unless one had something both new and tried to offer. Yet the subject has a fascination that increases with experience in operating, and to old-fashioned oculists—those who are capable of treating diseases of the eye as well as the errors of refraction—it is ever one of interest. The compiling of statistics by the great surgeons, the publication of results in 100, 500 or 1,000 consecutive operations, the recitation of accidents and of the means of treating and of preventing them, the dissemination of methods for the avoidance of mistakes, are a great boon to the followers of these surgeons and to the victims of cataract. But the record of failures and the causes of failures by men of large experience would be of more service to the beginner than that of their successes. Unfortunately for the oculist's peace and comfort, he does not bury his failures. They seem to live forever to tell their tales of woe to sympathetic hearers—oculists and others. Naturally we do not like to publish our mistakes, believing that the publicity given by the victims is quite enough. Yet the story is only half told if the failures are suppressed.

The causes of failures are many. Conspicuous among them stands the inexperience of the operator. With inexperience may be mentioned unsuitable personal habits and insufficient preparation. The beginner must qualify himself by many operations on animal eyes, standing or

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sitting in the proper relation to the eye, as though it belonged to a human subject, and acquiring accuracy and skilfulness in holding and manipulation of the instruments. Other requisites are good vision, steady hand, perfect illumination, coolness and judgment in difficulties and a full appreciation of his responsibilities. Perhaps the mention of the need of good vision on the part of the operator may seem absurd. It seems inconceivable that any man would approach an operation on the interior of the eye with vision insufficiently acute to give him perfect control of the situation; yet it has happened within my own experience and will happen again. Imperfect vision leads to nervousness and nervousness to disaster. When possible the illumination should be by means of a portable electric lamp of sufficient power, intelligently held by an assistant so that the light shall be reflected into the patient's eye and the surgeon's eyes protected. Thus the annoying corneal reflexes incident to daylight will be avoided. A steady hand is indispensable. If trembling exists its causes should be learned and removed. If it is stage fright the temptation to operate should be resisted until greater familiarity gained by observation of others and repeated practice on animal eyes assures the necessary composure. Intemperance in living, too much smoking or drinking must be controlled and one's habits so regulated that one's physical condition may be fit for the great responsibility assumed. No surgeon has the right to jeopardize the successful result of his operation by thoughtlessness and disregard of precautions. These statements, platitudes though they seem, are worth thinking about and acting on. We operate in sterile gowns, with sterile instruments, using sterile collyria, the field of operation sterile and the body protected by sterile sheets, carrying out with great precision all the regulations suggested in the canons of antiseptic surgery and yet ourselves go to the operating table unprepared and physically unfit.

For patients with uncomplicated senile cataract, long preliminary residence in a hospital or undue postponement of the time appointed for operation is both unnecessary and harmful, particularly to those who are nervous and despondent. The new environment, the consciousness of the impending operation, the isolation of blindness and the fear of the operation and its results induce baneful mental excitation. Twenty-four hours before operation gives sufficient time for thorough physical examination, including urinary analysis and antiseptic preparations; indeed, three or four hours may be not too short a time when one is familiar with the patient's state of health or has had the patient under observation.

DOES PRELIMINARY IRIDECTOMY CONTRIBUTE TO A SUCCESSFUL RESULT?

Operators who invariably perform simple extraction in uncomplicated cases disbelieve in the procedure of preliminary iridectomy for the assigned reasons that the dangers of two operations are greater than those of one, and that two should be avoided when one will suffice. To the unprejudiced mind this reason is not convincing. The real question is: Are the dangers of the simple or combined extraction less than those of extraction preceded by iridectomy? This subject was fairly presented to the Section by Reber in 1906. The majority of opinions collected by him favored preliminary iridectomy in complicated cataracts, in immature cataracts, and when the patient had but one eye.

A personal expression of opinion may be permitted. I prefer to perform preliminary iridectomy when all conditions permit, the patient's time, his degree of blindness or the length of time he may count on useful vision in the other eye, to a certain extent his social and financial status, his willingness to leave the decision to my judgment or, otherwise expressed, his confidence in me not only as an oculist but as a man who will advise for his patient's best interests. This statement of preference applies to the so-called uncomplicated cataracts, those cases in which most operators do not consider preliminary iridectomy. I believe it is the safest method of operating, else why is it preferred when the patient has but one eye? Iridectomy is a simple procedure and easy to perform. Under complete cocain anesthesia it is absolutely painless. Recovery is prompt and in most cases certain. Prolapse of the iris is almost never seen. Entanglement of the iris in the corneal wound is rare; the wound is small and closes in a few hours. If postoperative inflammation develops it is of milder grade than that following completed extraction. The subsequent extraction is simplified, a decided advantage to both patient and operator. For immature cataracts demanding operation preliminary iridectomy is positively indicated, and I can not agree with those operators who assert that as good visual results can be obtained by other methods, except possibly the extraction of the lens in its capsule.

During the past year I have performed preliminary iridectomy many times, extracting the lens three weeks to three months later. The results have been so satisfactory that I am convinced that the procedure is the operation of choice. I am not positive that the lens grows more rapidly opaque, but I have come to think that the operation may be classified among the ripening operations without their disadvantages. Haessig, whose experience has been very great, says: "Of the operation of extraction after preliminary iridectomy, every phase of the operation, the cystotomy, the expression of the lens, the expulsion of the cortical masses, can be performed without disturbance. The condition of the iris may be readily seen, there is less temptation to squeeze the lids, and undesirable complications—as loss of vitreous, eversion of the corneal flap—are seldom met.[1]

WHEN IS A CATARACT MATURE?

Technically the cataract is ripe when it is entirely opaque. For the purposes of operation (complete extraction) it is mature when the nucleus and the cortex are no longer distinct, but are united into one body, so that after corneal incision, well directed and accurately estimated pressure on the cornea will dislodge and expel the entire lens. In the pathology of cataract this stage is reached when "the lens finally breaks down into a pultaceous mass consisting of drops of fat, Morgagnian spheres, remains of lens fibers and an albuminoid liquid; fluid collects in separate vacuoles between the lens substance and the capsule of the lens, finally separating the capsule entirely from the cortex so that the lens can be shelled out of its capsule in cataract extraction."[2] Therefore, the time best adapted for operation is not when the entire lens is opaque, but when it is hard and its different parts adherent to one another and not to the capsule. The occasion of this final culmination of the formation of cataract varies greatly in different individuals and depends both on the underlying cause of the lenticular opacity and on the senility of the patient. Some boys reach maturity at an earlier age than others; some men are older than their years. It is stated in text-books that a cataract is soft before the patient is 25 and hard after 35 years of age. This implies that needling is the proper operation in the former, extraction in the latter. We are led to believe that after 55 or 60 at the latest the lens is hard enough to extract entire, whether opaque or transparent. The constitutional changes incident to advancing years must be considered in their application to cataract and allowance made for individualities.

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[1] Mittellungen ueber die letzten 500 Extract. 1908.

[2] Gibbon's Diseases of the Eye, 1905.

Miss A., aged 35. Preliminary iridectomy followed in one week by extraction. No lens debris. V. 20/70. Old corneal opacities.

Mr. S., aged 62, iridectomy in February, extraction in June. No complication in either operation. Lens matter in anterior chamber, chronic iridocyclitis and glaucoma.

Thus one patient, at an age long before the lens is believed to be hard enough to extract entire, recovered promptly and fully, and the other, beyond the prescribed age, had a bad result because the lens was not solid. The degree of opacity of the lens is not a substantial basis for judgment of its degree of solidity. I have removed without difficulty an almost transparent lens from the eye of a woman of 55, and I believe the experience is common to find as late as the seventieth year, lenses entirely opaque but with soft and tenacious cortices. My observation has led me to believe that in corpulent patients the lenses harden only very late, while in meager, emaciated persons the lens seems to partake of the sclerosis of other tissues of the body. The influence of intraocular disease in hardening the lens is problematic. That it induces opacity is well known, but the process is one of degeneration from malnutrition rather than a sclerosis. The answer, then, to the question, "When is a cataract mature"? resolves itself into the simple statement: The proper time for the extraction is to be determined by the degree of the loss of vision. Extraction may be performed at any age, the condition present in each individual case to decide the method adopted.

POINTS IN REGARD TO METHOD

Of the unavoidable causes of failure, such as intraocular hemorrhage, uncontrollable patient, coughing. disease of the eyeground, infection, postoperative delirium and accidents therefrom, it is beside the purpose of this paper to speak. The number of losses may be materially reduced by the operator's impersonal consideration of the qualities of character and temperament and the ocular and bodily health of the patient. For example, in the preliminary training, he may learn the chances of the patient's will or power to rotate the cornea downward. If these are unfavorable, general anesthesia should replace cocain anesthesia. It is my custom to assure my patient that if he will do as I tell him he will positively have no pain and, in all probability, a successful result, but that it is essential to success that I have his cooperation.

The speculum is left in place throughout the entire operation provided I see no indication of loss of vitreous, but in about 50 per cent. of cases I remove it after the capsulotomy. The indications are restlessness, pressure by the speculum on the ball, gaping of the wound and a sight of the vitreous in close proximity to the posterior lip. An assistant's services should be confined, in the case of obedient patients, to handing the instruments in their order to the operator. It is quite unnecessary for him to aid in the operation itself. Fixation is made during the section only; iridectomy and capsulotomy are more safely made without fixation. Expulsion of the lens is satisfactorily accomplished by pressure through the lower lid, and, as an accessory, if advisable. a spoon placed against the sclera posterior to the wound. The toilet is a matter of great importance. Its function is not only to restore the pillars of the coloboma but to cleanse the wound of lens matter. invisible shreds of capsule and clots of blood. Imperfect toilet means delayed union, cystoid cicatrix, and possibly infection. Lens debris not capable of removal by manipulation may be washed out by warm salt solution forcibly injected into the anterior chamber by a small-mouthed pipette, and if, after convalescence is well established, the anterior chamber still contains flocculi, dionin in increasing strength will bring about their absorption.

In conclusion, a word concerning imperfect local anesthesia—a prolific cause of accident. A 4 per cent. solution of cocain hydrochlorate applied freely to the conjunctival sac four times at intervals of four minutes, the lid closed after each instillation, insures complete anesthesia of the conjunctiva, cornea and iris. The corneal epithelium is not disturbed nor is the tension of the ball altered. The partial dilatation of the pupil is not a disadvantage; on the contrary, it contributes slightly to the facility of the performance of iridectomy and capsulotomy, and, in simple extraction, the passage of the lens through the pupil.

1528 Walnut Street.

MEMORANDA

THE EXPRESSION OF CATARACT IN ITS CAPSULE

WITH REPORT OF FORTY OPERATIONS *

H. V. WÜRDEMANN, M.D.
SEATTLE, WASH.

Delivery of the lens in its capsule has been an occasional accidental accompaniment of the cataract operation experienced by most operators ever since the beginning of cataract extraction, by reason of unwonted pressure on the globe by the operator or by the patient squeezing his lids (Case 10), during which more or less vitreous has been lost. It may also be occasioned knowingly, though unwillingly, by reason of rupture of the zonula after incision of the cornea when the operator has passed a spoon behind the lens lifting it out, or when he has premeditatedly removed a dislocated lens by the same means. Over-ripe and Morgagnian cataracts have occasionally likewise been removed by much the same procedure. Those of us who have been operating for a score of years will doubtless remember many such experiences and our surprise at the unusually good visual results obtained even in some instances in which considerable vitreous was lost. The deliberate routine extraction of all senile cataracts in their capsules was first introduced by A. Pagenstecher (September, 1865) at the Heidelberg Ophthalmological Congress and has been followed since that time by him and his brother,

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H. Pagenstecher[1] in a large proportion of their cataract operations.

Nach dem Drittelbogenschitt nach oben mit Bindehautlappen wird iridectomiert und hierauf die Linse, entweder durch das blosse Druckmanöver oder mit dem flachen Löffel der bloss als gleitfläche dient, entbunden.[2] **The lens is expelled after incision of about one-third of the circumference made with a conjunctival flap and an upward iridectomy, either by means of gentle pressure or with the flat spoon acting purely as a gliding surface.**

The above description of Pagenstecher's operation on cataract in the unopened capsule is as short and accurate as has yet been given. Examine these words closely, the various essays of Pagenstecher, digest them, then follow up by reading the essays of subsequent writers as Cassiani,[3] Gradenigo,[4] Gayet,[5] Landesberg,[6] and other foreign authors, Roosa,[7] Andrew,[8] Henry Smith,[9] Oxley,[10] Maynard,[11] Herbert,[12] Cheney[13] and

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[1] Pagenstecher, H.: Extraction de la cataracte sans ouverture de la capsule, Ann. d'ocul., 1871, lxvi, 126; Zur pathologischen Anatomie des Glaskörpers, Zentralbl. f. d. med. Wissensch., 1875, p. 708; Die Extraktion des grauen Staars in der geschlossenen Kapsel, Wiesbaden, 1877; Ueber Extraktion der Katarakte in der geschlossenen Kapsel, nebst Bericht über 117 Fälle, Arch. f. Augenh., 1881, x, 166; Ueber Staarextraktion mit ohne Entfernung der Kapsel, Arch. f. Ophth., 1888, xxxiv, 145; Ueber die Kataraktextraktion in der geschlossenen Kapsel, Ber. d. VII internat. Ophth.-Kong. zu Heidelberg, 1888, 506.

[2] Czermak, edited by Elschnig, A.: Augenärtzlichen Operationen, 1908, ii, 524 et seq.

[3] Cassiani, Ingoni: Dell' estrazione capsulo-lenticolare della cataratta, Ann. di ottal. 1897, p. 460.

[4] Gradenigo: Estrazione di cataratta capsulo-lenticolare perlscleram, Ann. di ottal., e lavori d. clin. ocul. di Napoli, 1902, xxxi, 702; De l'extraction de la cataracte dans la capsule par la sclérotique après zonulotomie, Ann. d'ocul., 1903, cxxx, 25.

[5] Gayet: Rapport sur l'opération de la cataracte, Ber. d. VII. internat. ophth. Kong. zu Heidelberg, 1888, p. 108.

[6] Landesberg: Ber. über 123 Staaroperationen, Arch. f. Ophth., 1878, xxiv, 59.

[7] Roosa: Remarks on the Extraction of the Crystalline Lens in its Capsule, Tr. Am. Jour. Ophth., 1885, ii.

[8] Andrew: The Operation of Senile Cataract in its Capsule, Brit. Med. Jour., January, 1883, p. 41.

[9] Smith, Henry: Indian Med. Gaz., June, 1900; Extraction of Cataract in the Capsule, Brit. Med. Jour., Sept. 26, 1903; Extraction of Cataract in the Capsule, Arch. Ophth., 1905, xxxiv, No. 6; Arch. of Ophth., March and May, 1906; Treatment of Immature Cataract, Arch. Ophth., November, 1908.

[10] Oxley: Cataract Extraction in the Capsule, Indian Med. Gaz., December, 1905.

[11] Maynard: Cataract Expression in the Capsule (Smith Operation), Ophth. Rev., 1906, p. 225; Manual of Ophthalmic Operations, 1908, pp. 106 et seq.

[12] Herbert: Extraction of the Lens in the Capsule, Ophthalmoscope, 1906, p. 113; Text-Book of Cataract Extraction, 1908.

[13] Cheney: Extraction of Cataract in the Capsule: Report of Ten Cases, Ophthalmology, October, 1906, iii, No. 1.

Greene,[14] then come back to the original of Pagenstecher and give credit for the devising of this operation in all its details where it belongs, not to later operators, even though of astounding skill, sufficiency and experience.

The recent revival from the antipodes has awakened new interest in this operation, largely occasioned by the astounding number of cases reported by Major Smith, of Jullundur, India, who has had an experience of about 20,000 operations, about 17,000 being in the capsule, among them many incipient cataracts.[9] While this vast experience has authoritatively established expression as successfully applied to the removal of senile cataract, yet the method should not be denominated "Smith's operation," even though he claims "the details of the operation are my own"[9] and has been accorded these claims in some quarters. In common with Major Herbert, I fail to see any substantial reason for such claims. "The operation is merely ordinary combined extraction, with the omission of one step—the opening of the capsule" (Herbert).[12] Smith insists that men must see it done in order to learn the method completely. By examination of the literature we "find no detail which has not been practiced earlier by others." "Those who have seen Smith operate do not appear to have benefited much, judging from their results" (Herbert).[12] I have not yet had the opportunity of seeing this master hand at work, but his writings certainly do not give us anything beyond the "omission of detail" (Herbert).[12]

I find nothing original except the use of the large squint hook for delivery of the lens, a minor and useful adjunct, but one which I have myself used since 1888, and probably many others have used a similar instrument for this purpose. A. and H. Pagenstecher have used a curved spoon since 1865. The large old-fashioned rounded strabismus hook is a perfectly adapted instrument for this purpose—its convexity indents the cornea properly and its concavity follows up the cataract until delivery through the open wound is completed, with much less force than by finger pressure through the lid, with more dexterity than by use of the Pagenstecher spoon, and with much less danger of bursting the hyaloid membrane and consequent prolapse of vitreous.

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[14] Greene: Extraction of the Cataract within the Capsule, Ophthalmology, January, 1907, iii, No. 2; Visual Results After Cataract Extraction, Ohio State Med. Jour., Sept. 15, 1906; Further Experience with the Smith-Indian Operation in the Extraction of Cataracts, Ophth. Rec., October, 1908; Tr. Sec. on Ophth. A. M. A., 1909.

Until I got the Smith hook I used a crochet hook which I devised in 1889 (but never reported) for ocular muscle operations. Last year (1908) I devised a large hook with a blunt crochet end. The latter is useful just as the lens is being delivered, to lift it out of the wound. Smith's new spoon is very like the old spoon of Pagenstecher; comparison of the two will not show any appreciable difference. In common with others I believed for many years that the insertion of the spoon behind the lens or into the vitreous back of the hyaloid membrane was the distinguishing mark of the Pagenstecher operation; but on referring to the original author and in correspondence with German writers I now fully agree with Elschnig (editor of Czermak's *Augenärztlichen Operationen*) in his letter to me of Feb. 1, 1909, in which he says: "Pagenstecher's operation is quite the same as Smith's."

Pagenstecher seems to have been the first to express the lens by external pressure (1871[1]) and Terson (1872[15]) and Wolkow (1894[16]) next report such procedure. Smith does not write until 1900.[9] Pagenstecher used his spoon to press the posterior lip of the wound back and to slide the lens thereupon. Smith[9] now passes the spoon behind the edge of the lens to assist its birth or extrusion, passing it into the vitreous on occasion of a hard delivery. So did A. and H. Pagenstecher and so did all of us.

The modifications of the Pagenstecher operation have varied from bizarre changes in place of section (Macnamara, outward, 1871[17]), introduction of spoon, hook or loop (Caspar, 1878[18] Higgens 1888[19]), tearing of zonula (Andrew, 1883[8]), dislocation of lens into an-

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15. Terson: De l'extraction du cristallin et de sa capsule, Rev. méd. de Toulouse, 1872, Nos. 3 and 4; Ann. d'ocul., lxvii, p. 313.

16. Wolkow: Ist die Kapsulotomie bei der Extraktion des Altersstaars nötige? Vestnik oftalmol., 1894, xi, 366.

17. Macnamara: A Manual of the Diseases of the Eye, 1871, ed. 11.

18. Caspar: Ueber Extraktion der kataraktosen Linse in geschlossener Kapsel, Inaug. Diss., Berlin, 1878.

19. Higgens: Extraction of Immature Cataract, Lancet, London, 1888, No. 19.

terior chamber by sclerotic puncture, Canstatt (1870[20]), Roosa (1885[7]) tore the zonule by partly dislocating the lens with the Graefe knife, Gradenigo (1895[4]) used a zonulotome, Wolkow (1894[16]) used two spoons for pressure and counter-pressure, etc. Mulroney, at Amritsar, India, began in 1890 to make a downward section without iridectomy, and this procedure has been adopted by Smith[9] (with upward corneal section), "who now has many imitators."[12]

We will now take up the operation itself according to my experience, leaving other phases and details to our confrère, Dr. Greene.

I do not deem the purely corneal section acceptable, for this is followed by slow healing, iridic prolapse (Herbert[12]), distorted pupils (Greene, 1906[14]) and excessive astigmatism, neither is the operation without iridectomy, on account of iridic prolapse. My experience has shown the 2/5 sclerocorneal section with conjunctival flap, as where this is made delayed union is unknown (Maynard[11]), combined with a very small iridectomy and extraction of the lens, *i. e.*, the regular operation less a capsulotomy, in immature and hypermature cataracts, is the only all-round satisfactory operation. The ordinary run of mature cataracts may be as well operated on with less risk in the average hands by the generally adopted combined flap extraction with capsulotomy; cataract in children and traumatic cataracts by discission.

The "absence of details," *i. e.*, the least handling of the eye and the fewest instruments possible, is the characteristic of this operation and what with the *tactus eruditus* makes it successful in the hands of Smith, Greene and others; while in some other hands—notably the critics of the method—especially those of India—"do not appear to have benefited much, judging from their results."[12] It is no operation for a beginner, neither is it for the average operator.

A stop speculum should not be used on account of the ever-present danger of vitreal prolapse. The lifting of the upper lid by a squint-hook seems crude. I use the lidholder of William Fisher, of Chicago, and have the patient's upper lid—the only one of moment—under

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20. Canstatt: Briefl. Mitteilung betreffend eine neue Idee sur operative Heilung des grauen Staars, Klin. Monatsbl. f. Augenh., 1870, pp. 131-133.

full control, and thus lose vitreous in not more than 5 per cent. of cases either with or without capsulotomy. The eye is fixed by catch forceps, the knife is entered (the only instrument entering the eye), and a corneoscleral section with a conjunctival flap is made. A very small iridectomy is made, the section of the iris only embracing the pupillary margin and edge of the sphincter muscle, by pressing the ends of iris forceps on the cornea over the upper part of the pupil, when the iris will usually prolapse, the edge being seized by the forceps, the iridectomy is completed by a single cut of the scissors. The bend of a large hook is then pressed moderately, slowly and continuously on the cornea at the lower edge of the lens, that tilted backward, the pressure gradually relaxing as the upper edge of the lens enters the wound, the body of the lens is lifted up by the pressure of the hook, which follows it in its delivery; the flat spoon of Pagenstecher or Smith may be used to assist the lens to slide out of the eye by offering an inclined and smooth plane for it to glide on. Seldom is the spoon entered into the eyeball; forceps are occasionally necessary to seize the lens when impacted between the lips of the wound; and the act is completed by the crochet end of the hook catching the lens, on its delivery, rotating it out of the wound, the conjunctival flap and the iris stroked back into place, the lids closed and a light bandage applied. This is a brief description of the method I have adopted and adapted. My expression cases are comparatively few, but compare very favorably, and in fact the results are to my mind better than those of about 1,000 regular extractions performed by me within the last twenty years.

My cases, grouped under dates of the expression operations, are as follows:

CASE 1.—Oct. 3, 1904: Mrs. S., aged 58. Immature cataracts R. & L. Preliminary iridectomy and direct trituration with Boettmann's spoon by me October, 1903, on L. E.; no ripening. Extraction of cataract with aid of Pagenstecher's spoon which entered the eye, no loss of vitreous. V. + 9.00 $\substack{\frown \\ \smile}$ 1.00, 180° = 6/vi.

CASE 2.—Oct. 15, 1905: Same as Case 1. I had done preliminary iridectomy without trituration on this eye in October, 1903. Normal expression operation on mature cataract of R. E. V. + 9.00 $\substack{\frown \\ \smile}$ + 1.50, 165° = 6/vi.

Case 3.—March 12, 1906: Mr. F. D., aged 65. R. immature, L. mature. Small iridectomy, normal expression in capsule of L. V. + 12.00 $\overset{\frown}{\smile}$ + 2.00, 90° = 6/xii. See also (12).

Case 4.—March 9, 1907: Mrs. Z., aged 61. Hypermature black cataract, small iridectomy, expression with slight loss of vitreous, corneoconjunctival stitch after. V. + 10.00 $\overset{\frown}{\smile}$ + 3.00, 180° = 6/xxiv.

Case 5.—March 18, 1907: Mrs. S., aged 59. Mature cataract, small iridectomy normal expression. V. + 10.00 = 6/xii.

Case 6.—March 23, 1907: Mrs. E. J. J., aged 60. Mature black cataract, small iridectomy, expression normal until lens was pinched in wound when a portion of the anterior capsule tore off, sticking in wound, removed by forceps without entering the eye, a slight iritis developed; the hyaloid membrane apparently participating in the opacifying process, as several months afterward a fine membranous opacity was observable on focal illumination. I have not seen this occurrence before or since. V. + 10.00 $\overset{\frown}{\smile}$ + 150, 15° = 6/vi; but on development of the hyaloid opacity was reduced to 6/viii with correction.

Case 7.—April 10, 1907: C. C., aged 48. Mature cataract, small iridectomy, normal expression. V. 10 days later with 10.00 about 6/xxiv. I did not have the opportunity of prescribing lenses or observing the vision later as he did not return after leaving the hospital.

Case 8.—June 19, 1907: B. O., aged 65. Mature cataract, normal expression except capsule ruptured, lens removed by sliding on spoon and capsule pulled out of wound with forceps without entering the eye. V. + 10.00 $\overset{\frown}{\smile}$ + 2.50, 180° 6/viii.

Case 9.—Nov. 6, 1907: R. W., aged 45. Immature cataracts, small iridectomy, normal expression, corneal incision (the typical Indian operation), very slow healing of wound, iritis, four weeks in hospital. High astigmia followed, although visual acuity was very good. V. + 11.00 $\overset{\frown}{\smile}$ + 4.00, 135° = 6/xviii.

Case 10.—Nov. 11, 1907: Mrs. A. S. Hypermature cataracts with tremulous irides and synchysis vitrei. There being fair perception and iris reaction in the left, I attempted ordinary combined extraction, corneoconjunctival stitch (Kalt) placed before incision, but lens popped out in the capsule with considerable loss of vitreous. Result for a few months was V. = movements of hands and large objects. Further degeneration set in so that in May, 1908, eye was blind. (See Case 19).

Case 11.—Nov. 12, 1907: Mrs. S., aged 70. Hypermature cataract, small iridectomy, capsule ruptured and was not entirely removed from eye on account of rupture of hyaloid with slight loss of vitreous, caused by attempt at removal by iris forceps entering the eye, conjunctival stitch. Four weeks in hospital with iritis. V. two months afterwards + 12.00 = 6/xxxvi.

Case 12.—Dec. 13, 1907: Patient in Case 3 returned on account of satisfactory result on L. E.; V. then being 6/viii with old correction, increased to 6/vi + 2 with decreased cylinder. Normal expression, immature cataract, small iridectomy. V. + 12.00 ⊃ + 1.00, 90° = 6/viii.

Case 13.—Jan. 6, 1908: C. C., aged 40. Morgagnian cataract, normal expression, small iridectomy, lens aided in delivery by forceps as it became impacted into wound, no entrance of forceps. V. good. Refraction not recorded.

Case 14.—Jan. 29, 1908: C. W., aged 69. Immature cataracts both eyes. V. 6/lx and 3/lx. L. E., corneal incision, small iridectomy, normal expression except that lens came out very slowly and seemingly took considerable pressure in delivery. Striped keratitis for two weeks. Very high immediate corneal astigmia (6.00, 180°). V. + 11.00 ⊃ + 5.50, 180° = 6/xxxvi. Astigmia reduced in May, 1908, to 3.00, 180°.

Case 15.—Feb. 21, 1908: E. B., aged 58. Immature cataract, rather large iridectomy, expression followed by small bead of vitreous, conjunctival stitch. V. + 10.00 = 6/xii.

Case 16.—March 21, 1908: O. P. H., aged 75. Robust health, two years blind with mature cataracts. R. E. chronic conjunctivitis and dacryocystitis. L. E. apparently normal, no pus germs on microscopic examination, three days' bichlorid of mercury treatment to eye and Seiler's solution in nose. Small iridectomy normal expression, brown cataract. Corneal astigmia 1.50, 180° V. + 10.00 = 6/xxiv. Increased in May, 1908, to + 11.00 = 6/xii. (See Case 18).

Case 17.—May 5, 1908: Mrs. S., aged 64. Mature cataract, normal expression with rather large and uneven irridectomy. V. + 11.00 ⊃ + 1.50, 15° = 6/viii.

Case 18.—May 21, 1908: Patient in Case 16. April 1, 1908, I excised the lacrimal sac according to Meller's[21] method securing union by first intention and kept him under antiseptic treatment with bichlorid of mercury solutions locally until May 22, 1908, when I made normal expression of the right cataract with small iridectomy. V. + 10.00 = 6/xxiv.

Case 19.—May 21, 1908: Patient 10 returned with L. E. blind from degeneration, and as a last resort I made expressive operation on the R. E., which only had poor perception and projection, Morgagnian cataract, synchysis vitrei, having no complications and to my surprise getting 6/lx vision with + 10.00, but a few months later vision further deteriorated and the end results are unknown.

Case 20.—June 5, 1908: Mr. S., aged 43. Morgagnian cataract, normal expression, small iridectomy. V. + 10.00 = 6/lx.

Case 21.—June 19, 1908: Mrs. H. W., aged 61. R. mature L. immature cataracts, normal expression with small iridectomy R. except that capsule ruptured within the eye and

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21. Meller: Ophthalmic Surgery, 1908, p. 1 et seq.

some remained. Opacity cleared up under dionin. V. + 11.00 = 6/xxiv. (See also Case 28.)

Case 22.—June 20, 1908: Mrs. M., aged 60. R. E. blind from previous iridocyclitis following attempted extraction of cataract by another operator. L. E. poor perception and projection. L. E. Morgagnian cataract expressed aided by forceps which entered the eye, much loss of fluid vitreous. Cataract with black nucleus. No result as eye went on to iridocyclitis.

Case 23.—July 16, 1908: M. O., aged 63. Immature cataract, expression with small iridectomy, capsule ruptured before delivery, extracted with forceps entering the anterior chamber, small bead of vitreous extruded and was cut off, conjunctival stitch. V. + 10.00 ⌒ + 1.75, 180° = 6/viii.

Case 24.—July 21, 1908: N. O., aged 69, had an attempted extraction done by another surgeon in L. E. with result of incarceration of iris and partially dislocated immature cataractous lens with resultant vision of fingers at 2.00 M. As the vision of the R. E. was less than the L. E. through the lenticular opacity I attempted expression with iridectomy, but the capsule ruptured and vitreous escaped, necessitating entrance of the spoon and delivery of the lens, conjunctival stitch. Patient had postoperative mania occasioned by fear of not regaining vision, owing to his previous experience and from the fact that his brother was totally blind after an unsuccessful cataract operation by the same operator on his only remaining eye, the other being injured and blinded by an accident. Nov. 6, 1908, I made a discission of pupillary membranes securing a clear pupil with normal looking fundus; but patient declared and persisted in the statement that he could not see, becoming very childish, so he was taken home and the end-results are unknown to me.

Case 25.—July 30, 1908: Patient in Case 23 returned and I made normal expression of immature cataract with small iridectomy on left eye. V. + 10.00 ⌒ + 2.00, 180° = 6/vi.

Case 26.—Aug. 3, 1908: Mrs. M. S., aged 51. Immature cataract, normal expression with small iridectomy. V. + 10.00 ⌒ + 1.00, 180° = 6/xxx. As the other eye has yet good sight she prefers to go without this correction.

Case 27.—Aug. 6, 1908: Mr. F., aged 54. Immature cataract, normal expression with small iridectomy. V. + 12.00 ⌒ + 2.00, 15° = 6/xxxvi.

Case 28.—Sept. 2, 1908: Patient in Case 21. Expression L. with small iridectomy; fluid vitreous escaped, spoon entered eye, conjunctival stitch, hyphema, dionin. Oct. 22, 1908, discission. V. + 12.00 = 6/xxiv.

Case 29.—Sept. 18, 1908: J. R. W., aged 80. Mature cataract both eyes, very smooth normal expression with small iridectomy R. E. Clear black pupil and counting of fingers immediately after operation. Very nervous patient; two days

TABLE ILLUSTRATING FORTY OPERA

No. of Cases.	Type of Cataract.					Visual Acuity.			Compli	
	Mature.	Immature.	Hypermature.	Morgagnian.	Complicated.	Good Results. Vision 6/vi to 6/ix.	Poor Results. V = Objects.	Failures.	Infections.	Iridocyclitis.
....	..	1	..	..	..	1	..	..	..	..
2.......	..	1	..	..	..	1	..	..	..	..
3.......	1	..	..	..	..	1	..	..	..	..
4.......	..	..	1	..	..	1	..	..	..	..
5.......	1	..	..	..	..	1	..	..	..	..
6.......	1	..	..	..	..	1	..	..	..	..
7.......	1	..	..	..	..	1	..	..	..	..
8.......	1	..	..	..	..	1	..	..	..	..
9.......	..	1	..	..	..	1	..	..	..	..
0.......	..	..	1	..	1	..	1	..	..	1
....	..	..	1	..	..	1	..	..	..	..
2.......	..	1	..	..	..	1	..	..	..	..
3.......	..	..	..	1	..	1	..	..	..	..
4.......	..	1	..	..	..	1	..	..	..	..
5.......	..	1	..	..	..	1	..	..	..	..
6.......	1	..	..	..	..	1	..	..	..	..
7.......	1	..	..	..	..	1	..	..	..	..
8.......	..	..	1	..	1	..	1	..	..	..
9.......	1	..	..	..	1	1	..	..	..	..
0.......	..	..	..	1	..	1	..	..	..	..
1.......	1	..	..	..	..	1	..	..	..	..
2.......	..	..	..	1	1	..	..	1	..	1
3.......	..	1	..	..	..	1	..	..	..	..
4.......	..	1	..	..	..	..	..	1	..	..
5.......	..	1	..	..	..	1	..	..	..	..
6.......	..	1	..	..	..	1	..	..	..	..
7.......	..	1	..	..	..	1	..	..	..	..
8.......	1	..	..	..	..	1	..	..	..	..
9.......	1	..	..	..	..	..	..	1	1	..
0.......	..	1	..	..	1	1	..	..	..	..
1.......	1	..	..	..	1	1	..	..	..	..
2 to 37.	3	3	..	..	..	6	..	..	..	..
8.......	1	..	..	..	1	..	..	..	..	..
39.......	1	..	..	..	..	1	..	*	..	..
40.......	1	..	..	..	1	..	..	..	..	..
	18	15	4	3	8	34	2	4	1	2

* 1 ultimate detached retina.

ESSION OF CATARACT IN ITS CAPSULE.

ealing.		Gen'l Comps.	Vitreous.		Capsule.		Inst. Enter'g Eye.		Incision.	
Hyaloid Opacity.	Delayed Union and High Astigmia.	Post-op. Mania or Psychic Disturbances.	No loss.	Loss.	No Rupture.	Rupture.	Graefe Knife Only.	Spoon, Loop or Forceps Beside Knife.	Sclero-corneal with Conjunct. Flap.	Corneal.
..	..	..	1	..	1	..	..	1	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	..	1	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
1	..	..	1	..	..	1	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	..	1	1	..	1	..
..	1	..	1	..	1	..	1	..	..	1
..	..	..	..	1	1	..	1	..	1	..
..	..	..	..	1	..	1	..	1	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	..	1	1	..
..	1	..	1	..	1	..	1	..	..	1
..	..	..	..	1	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	1	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	..	1	..	1	1	..
..	..	..	..	1	1	..	..	1	1	..
..	..	..	..	1	..	1	..	1	1	..
..	..	1	..	1	..	1	..	1	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	..	1	..	1	..	1	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	1	..	1	..	..	1	1	..
..	..	..	1	..	1	..	1	..	1	..
..	..	..	5	1	4	2	3	3	6	..
..	..	..	..	1	1	..	..	1	1	..
..	..	..	1	..	..	1	..	1	1	..
..	1	..	1	..	1	..	..	1	..	1
1	3	2	30	10	30	10	25	15	37	3

later had postoperative mania, got his fingers under the bandage despite attention of special nurse, infected eye which was lost through panophthalmitis.

Cases 30 and 31.—Nov. 21, 1908: H. B., aged 57. Five years before this date I had extracted an immature cataract from his right eye, finding the zonula weak; the lens came out with the capsule. V. + 10.00 = 6/vi. Nov. 10, 1908, I attempted operation, but on completing sclerocorneal incision found that a bead of vitreous protruded into the anterior chamber, closed up the eye and November 21 made new incision with iridectomy; perfectly normal expression. V. + 10.00 ⊂ + 1.00, 180° = 6/viii.

Cases 32 to 37.—In addition to the above I have made six expression operations at my public clinic, the case histories of which are not complete nor at present accessible. In one of them there was loss of vitreous, in two rupture of the capsule but all recovered with useful vision.

I append three more recent case histories which apply to this subject, although they can not be classed as expression operations, in two of them the lens being removed in its capsule by the aid of the wire loop, in one of which detachment of the retina followed later, and in the other owing to the corneal incision a high degree of astigmia with delayed union followed the operation. In the other the interest centers on the failure of expression, followed later by successful combined extraction.

Case 38.—May, 1897: J. M. C., aged 50. A diabetic and myope of — 4.00 D. submitted to a flap extraction by me in the right eye. On account of rupture of the zonule and loss of vitreous I removed the lens by the wire loop, ten days later, V. with + 8.00 = 6/vii. Gradual loss of sight followed, and when seen a year later detachment of the retina and optic nerve atrophy was found.

Case 39.—May 2, 1908: Patient in Case 38. I made typical flap extraction with iridectomy on the left eye after attempting expression but had to do a cystotomy; the lens capsule, however, was extracted with forceps. V. + 10.00 ⊂ + 2.00, 90° = 6/xxiv.

Case 40.—September, 1908: I operated at a clinic on a man of about 60 in whom an attempt by another operator at extraction of cataract had been made but the lens had not been removed. Here I found a cataract with posterior synechiæ. Ordinary attempts at extraction of the lens after the incision of the sclerocornea without conjunctival flap failing to deliver I passed the wire loop behind the lens and delivered it without loss of vitreous. I understand on good authority that there was slow healing of the wound which possibly may have reopened and the lips of the wound overriding each other being followed by a very high degree of astigmatism 6.00 or 8.00 D. I have, however, not had the opportunity to examine the case since the day of operation.

I am convinced that from the circumstances under which the Indian operators are placed, the apparent lessened susceptibility to infection of their patients, the fact that wounds on Hindoos, like those on the lower races and wild animals, are more prone to heal than in the cultivated classes, and, above all, that these patients disappear from observation soon after the operation, we must depend for the end results on the testimony of others who practice in more civilized countries, where the patient's life history can be followed with more exactions. Therefore, the reports of Cheney,[13] Greene and others, although in proportion showing such a paucity of patients, are of more value to us.

"If the dread of the remote complications is relieved by further experience there is much to be said for the operation" (Elliot[22]). If the ultimate results of the operation are good in the foregoing cases I shall continue to practice it in selected cases.

The operation is not one to be attempted by any but the most skilled operator, and even with him is not to be chosen as a routine method, for it is a far more dangerous operation, except in the hands of an operator whose touch is fine, who has no trembling fingers and whose judgment is the most exact.

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[22] Elliot: Cataract Extraction, Brit. Med. Jour., Dec. 19, 1908.

MEMORANDA

EXPERIENCE IN THE EXPRESSION OF CATARACTS IN THE CAPSULE BY THE SMITH METHOD

[WITH A DEMONSTRATION OF THE STEPS OF THE OPERATION ON A MODEL OF THE EYE] *

D. W. GREENE, M.D.
DAYTON, OHIO

When Major Smith's article[1] on the extraction of cataract within the capsule by external manipulation appeared I became interested in the possibilities of the method and gave it such trial as I was capable of at that time in twenty-two cases. These were hastily brought together and not selected. The results were thought to be not satisfactory at the time and were so reported,[2] but further observation has shown that a very fair percentage of the patients did well. In the beginning it should be stated that I have only included in these later statistics cases of operation in which I have supervised the after-treatment and determined the refraction.

Of all the operations which have been proposed for the removal of cataract, no one of them and no combination of them offers such a complete and satisfactory disposition of the capsule, which is the beginning, middle and end of most of our after-troubles, as extraction within the capsule.[3] This is so evident that if any

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

1. Arch. Ophth., June, 1906.

2. Ophthalmology, January, 1907.

3. In connection with this statement the following articles will be found of interest: Capsular Complications After Cataract Operations, Brit. Med. Jour., 1905, p. 433; Knapp: The Lens Capsule in the Operation of Cataract, Tr. Am. Acad. Ophth. and Oto-Laryngol., 1905, p. 4.

added risk is incurred in doing the operation it seems more than offset by what is gained in the comparative freedom from the complications which are liable to follow the regular operation. Regarding these complications, Knapp long ago called attention to the fact that iridectomy alone was a comparatively safe procedure, but that opening the capsule and extracting the lens added other elements of danger to the traumatism of the iridectomy, and changed the condition to one of danger to the eye in a certain proportion of cases. These elements of danger consisted in the mechanical irritation of the lens débris to the cut and torn surface of the iris.[4] Hess and Schirmer regard this irritation as due to infection.[5] "It seems more than probable that the inflammation is due to the chemically altered lens substance which acts as an irritant, but whether it is trauma, infection or chemical irritation, it is generally acknowledged that the postoperative dangers—iritis, cyclitis or iridocyclitis—are in some manner attributable to the cortical débris."[6] If the true explanation of the causes of these postoperative inflammations is to be found in either of these theories, such sequelæ should not follow delivery in the capsule, and experience shows that delivery in the capsule furnishes a high degree of immunity from them. The combined operation, in the hands of the best operators of this and other countries in 1,032 operations, according to Ring, furnished a loss of vitreous in 7.23 per cent., iritis in 13.15 per cent., incarceration of iris in 4.32 per cent., and discission was required in 26.61 per cent.[7] According to Smith's statistics three years ago, in 2,616 operations he only had 6.8 per cent. of loss of vitreous and 0.3 per cent. of iritis in 2,494 operations when the lens came away whole, but when the capsule was opened intentionally or ruptured in delivery, and had to be left behind, iritis was observed in 5 per cent. of the cases.[8] Dr. Arnold Knapp, after a visit to Major Smith's clinic at Jullundur in 1906,[9] practically confirmed these statistics. We must, therefore, accept these percentages and seek their explanation in the marvelous skill of the operator and the comparatively early age at which cataracts develop in India. Cases of cataract, I understand, develop in persons between five and ten years younger in India than in this country. The degenerative changes of the vitreous are more marked in the ten years between sixty and seventy than at any prior period, and the condition of the vitreous is the essential all-important element which influences statistics as to its loss, the shape of the pupil, iris entanglements, etc., in the operation.

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[4] Arch. Ophth., xii, 98.

[5] Wooten, in Claiborne's Cataract Symposium, p. 163-164.

[6] Stricken: Innutrition of the Lens in its Relation to Cataract Formation, Ann. Ophth., viii, No. 4.

[7] Med. Rec., Feb. 23, 1905.

[8] Arch. Ophth., xxxiv, 601.

[9] Arch. Ophth., January, 1908.

Attention is called to the fact that the average age of my patients has been high (67.7 years). Largely because of this I think my percentage of vitreous loss has been high also. In making these later operations I had the advantage of having seen Smith perform it; I have learned by experience that the percentage of success depends more on the attainment of a high degree of skill, technical knowledge and the imitative faculty than on any skill acquired in making the regular operation. Theoretical considerations avail little or nothing in considering its place among cataract operations. The operation can be learned only at the elbow of one who is capable of demonstrating it. Without having this advantage and experience in the after-treatment of cases, no one is capable of passing judgment on the merits or demerits of the operation.

In July of last year, through the courtesy of Dr. Arnold Knapp, I saw Major Smith make three operations in New York City. So large a crowd was present that my view of the operations was not satisfactory, but I afterward spent several hours with him listening to descriptions of his method and seeing him demonstrate it until I thought I had a fair understanding of his technic. Major Smith regards the operation as in process of evolution. I quote from a late letter: "When once we have convinced ourselves that we are right, and have the doggedness to go ahead, our ultimate success is assured." Again he says: "You can not teach a man to do this operation by mere demonstration. He must do the operation before the teacher sufficiently often to acquire confidence in himself; and, further, a trained assistant who can properly expose the eye and at the same time take off all pressure is scarcely less important to the success of the operation than the operator himself." In this connection his address[10] before the American Ophthalmological Society at New London, Conn., last July, should be read. After describing the operation, he says:

> The treatment of immature cataracts which I have practiced extensively, and which I am an ardent advocate of, which I regard as the operation of the future, which will leave senile cataract a much rarer condition than at present, is extraction of the lens in the capsule, a procedure to which immature cataracts are admirably suited. I hold that, if for no other reason than for the treatment of immature cataract, every ophthalmic surgeon should be familiar with the art of extracting the cataractous lens in its capsule.

I submit with diffidence that three years ago I called attention to these same points in the treatment of immature cataract,[11] and further experience has confirmed my belief that the method is superior to any other method of extracting such cataracts, or to any method of artificial ripening. It is in the treatment of immature cataract that I think the Smith operation will receive its first recognition, because, if I understand the opinion of the profession on this point, it is not favorable to extracting unripe cataracts by regular methods. Major Smith has told me that just this line of reasoning and observation with capsulotomy methods and secondary operations and accidental deliveries in the capsule are what led him to seek a method of delivering all kinds of cataracts, which would at the same time remove the capsule with the lens, because he believes such disposition of it to be good surgery.

While writers from the time of Pagenstecher to the present have considered hypermature cataracts specially suited for removal in the capsule, my experience has been that these are usually the most difficult to express. The vitreous is often fluid, the strength of the zonula, while usually frail, can not be foretold, and the milky cortex will be forced by the pressure to occupy one end of the capsular bag and the small disc-like nucleus will float to the other. I have seen this hour-glass appearance several times. These are the cases which require careful handling, since they furnish a high percentage of loss of vitreous, but the same would be true if extractions were done by regular methods. In this connection we should remember that cataractous eyes are always diseased eyes, as Risley and others have so well shown. While it is my desire to discuss the operation from my personal experience, I recognize the difficulty of placing my experience, which is limited, but, on the whole, *is favorable to the Smith operation,* in opposition to the theoretical considerations which have influenced men against delivery in the capsule, and for comparison with the vast number of reports of regular operations and published statistics by innumerable observers there are no extensive statistics of delivery in the capsule available. The fact that possibly but one operation in the capsule to five thousand or more by capsulotomy methods has been made, ought not, *per se,* to prejudice men against accepting a new procedure which, to say the least, is theoretically desirable, because from the beginning the desire of the profession has been for a method which would remove the capsule with the lens. All have recognized the undesirability of leaving it behind and the surgical incompleteness of all operations which do so. Witness the early efforts of Richter and Beer, the later efforts of Sperino and de Wecker, and the still later method of Pagenstecher and Macnamara to accomplish this end. Loss of vitreous, as a rule, in my experience, has been more the fault of the patient than of the operation. This statement does not help the matter, but it puts the blame where it usually belongs.

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[10] Tr. Am. Ophthal. Soc., 1908; Arch. Ophth., November, 1908.
[11] Ophthalmology, January, 1907.

The fear of retinal detachment from loss of vitreous seems to be the *bête noir* of all men with whom I have discussed the operation, yet this danger is not supported by the available statistics, and it really seems to be very small. Pagenstecher more than forty years ago, in 600 operations, saw only one case of detachment of the retina, and that in a highly myopic eye with chorioiditis. Wetzel and Beer long ago stated that they had not seen harm resulting from a loss of one-third of the vitreous (by volume). Smith has stated that in his experience the same is true. Captain Lister of the Indian Medical Service, a former pupil of Major Smith's, has examined 100 cases of from one to seven years after loss of vitreous. He read a report at the Bombay Medical Congress in February; his conclusions have not yet been published, but Smith has stated that Lister did not find a case of detachment of the retina among them. It must, however, be admitted in this connection that loss of vitreous is looked on by the majority of men in this country with apprehension. Whether they overestimated its importance or not, time and observation alone can tell. I myself have seen only two cases in which vision following the operation merely equaled perception of light, which could be attributed to detachment of the retina after the loss of about one-half of the vitreous. In these cases there was delayed healing, gaping of the wound and an ingrowth of corneal epithelium, and reduction in size of the cornea in healing, so that no view of the fundus could be had. I have not been certain as to the cause of loss of vision in these cases because P. L. remained. Several times after a slight loss of vitreous I have smoothed out the iris and have seen many cases in which it would be impossible to tell that the lens had been removed in the capsule. On the other hand, in a few instances I have bandaged up an eye with a keyhole pupil and have found the wound open, vitreous presenting and the iris entangled in the angles at the first dressing. The same condition is observed after the regular operations. These conditions are usually observed in nervous and restless patients with high blood pressure. Loss of vitreous should not be the sole measure by which this operation is judged; if it is as serious as many think, the danger is yet to be proved. The statistics we have do not prove the danger to be great, and Smith's statistics show that with select cases it is possible to reduce it to the percentage of the regular operation or even below it. Surely comparative freedom from inflammation and high-class visual results count for more than slight loss.

Perhaps of more importance to the future integrity of the eye than loss of vitreous is the fact that no discission is necessary. It requires no argument to show that the risk of an operation increases in proportion to the number of times a knife-needle or scissors are passed into the eye. This is also one of the essential points on which those favorable to the Smith operation based their contention, *that it is both safer and more complete than the regular operation,* even if the loss of vitreous is greater for the average operator, *which must be conceded.* This liability to accident seems more than offset by the comparative infrequency of the reactions that are liable to follow a discission. Having had a large experience with extraction and after-treatment with capsulotomy methods, I have not seen such smooth and uncomplicated healing, all things being equal, in any other series of cases; there can be no question as to the correctness of this observation, and it is in harmony with the experience of all who are familiar with delivery within the capsule. On this point I do not wish to be misunderstood. I have encountered certain reactions, but they have not been of an inflammatory character (except in the few cases shown in the statistics), but have been connected with the healing of the section and have usually been associated with ruptures .of the capsule, incarceration of iris, or cystoid healing, and blood pressure above 160 mm. Hg. in nervous and restless patients. Only two cases of iritis sufficient to cause pain and require treatment have been observed. Both followed rupture of the capsule. A few times I have observed the intrusion of a bead of vitreous into the section at the first dressing, or even later when none had presented at the time of operation. It has not been sufficient as a rule to abscise it because more will take its place, but I have always secured firm union by repeatedly tapping the chamber below, and the same practice has been successful in cystoid healing after excising the cyst.

My experience with the operation has been largely with patients of advanced years in my clinic at the National Military Home, at St. Elizabeth's Hospital, this city (and in private practice) in whom the conditions of the vitreous and fundus are frequently unfavorable to high-class results.

I pass rapidly over the corneal incision, which should be an enlarged de Wecker without a corneal flap. I have tried making a large iridectomy, and have found it to have some advantages in the delivery of the lens, but, on the other hand, as the tendency is in the direction of too large a pupil, I have made a small iridectomy as a rule. The patient can look down during the completion of the section and the iridectomy, but during the expression of the lens must look straight ahead or slightly upward, *never downward.* I have seen more loss of vitreous from the patient violating this injunction than from any other cause.

Smith's late manipulation with the end of the spatula and strabismus hook, over the lower third of the lens, by which it is dislocated, has been the most difficult step to imitate. I am conscious that failure to make this manipulation properly has been the weak point in my technic, and has materially increased my percentage of vitreous loss. I have dispensed entirely with the speculum while making the section and the iridectomy and have had my assistants use the Fisher and later the Noyes lid-elevator instead. The assistant changes to the large hook after the iridectomy, and lifts the upper lid as Smith has directed. The operator should make light or moderate pressure to and fro over the lower third of the lens with the tip of the strabismus hook. At this stage the operator will see the lower border of the lens mapped out through the cornea and iris advanced slightly, then with moderate pressure with the small end of the spatula applied edgewise across the lower sclerocorneal border the lens will advance still farther toward the incision, the zonula will give way, and the top of the lens will often tilt slightly backward. If the pressure is continued around the lower border of the lens, and the tip of the hook inserted under or behind it, the intraocular pressure will aid in causing the lens, which is now only anchored above, to describe a half-circle. The wound will gape, and the lens will emerge bottom edge upward, leaving only a small band of zonula to be loosened. This must be done slowly and with the utmost gentleness by drawing it back and forth through the incision. Otherwise the capsule will rupture and recede into the eye. It is still possible to catch it with forceps if the operator be dextrous. Vitreous is seldom lost when the lens is delivered in this way. In more than half of these cases the lens has been delivered thus. In other cases the zonula will rupture first at one of the angles of the section; then if the vitreous is fluid some of it is easily lost. It is at this point that familiarity with what may happen and skill and technic in meeting the emergency obviates some mishaps.

Before leaving these steps in operation I wish to call attention to the perfect control the assistant has over the orbicularis with the large strabismus hook under the upper lid. Slight traction with it will break the power of the muscle by bending it at a sharp angle. With a small piece of gauze on the face of his thumb the assistant pulls down the lower lid. With a trained assistant it is practically impossible for the patient to squeeze out normal vitreous. Unfortunately not all patients have normal vitreous and it is easy for the patient to lose fluid vitreous not only by attempts at squeezing but by suddenly and forcibly looking down. So far as I am able to learn, the particular design of this large-hook, and the use to which it is put, the control of the lower lid in the way described and the use of the smaller hook for delivering the lens, must be credited to Major Smith. To his credit also, I think, belong the manipulation in emergencies with the double-ended spatula, one end of which is passed through the vitreous presenting behind the lens. It should not be passed deeper than the posterior pole of the lens, while pressure through the cornea below will cause the lens in its capsule to glide up this inclined plane to its exit. In my judgment these three instruments are the best additions that have been made to the armamentarium of the ophthalmic surgeon in many years. Since I have been making more pressure with the tip of the spatula and done less rubbing over the lower third of the lens the number of cases of striped and latticed keratitis have markedly diminished, and I have not seen either remain and lower vision in any of these cases. I never use bichlorid solution in cataract operations; therefore it could not be responsible for the keratitis. There are no rules governing the amount of pressure to be used in delivering the lens. Each case is a law unto itself. Three points should be remembered: 1. The pressure should never be excessive, but steady and equal. 2. The cornea should be kept moist. 3. There should be no pressure or rubbing over the center of the cornea.

It is in connection with these manipulations that men become prejudiced against the operation when they see it performed for the first time, because they overestimate the force of the steady pressure required to rupture the zonula and dislocate the lens, and consider the manipulations rough and unscientific. I have satisfied several who have visited my clinics that the degree of pressure used is not great, but is much more apparent than real. In other words, they have unlearned some of their misconceptions regarding this important step of the operation.

The lens having been delivered, the iris, if prolapsed, should be returned and smoothed out, but the danger of loss of vitreous should not be forgotten in doing this.

TABLE OF RESULTS OF SMITH OPERATION IN 75 CASES.

No.	Age.	Eye.	Condition of Cataract.	B. P.	Lens Delivered.	Capsule.	Vitreous Loss.	Iris.	Chamber Formed.	Vision.	Remarks.
1	73	L.	Im.	140/100					3d day.	20/20	
2	71	R.	M.	190					14th d..	20/30	Presenting vitreous; tenth day passed knife through wound; firm union.
3	61	L.	M.	190					3d day.	20/20	
4	60	L.	M.	210/130		Ruptured, retained.		Iritis.	14th d..	20/20	Discission.
5	75	R.	Hm.	160/120					3d day.	20/20	
6	68	R.	Im.	190/120					4th d..	20/30	
7	49	R.	M.	120/100					2d day.	20/15	
8	76	R.	M.	200/130					3d day.	20/20	Wound opened after 1 week. T = + 1: chamber formed next day.
9	60	L.	M.	190/130	Spatula..		Slight		4th d..	20/70	Myopia and chorioiditis.
10	65	L.	Hm.	165/130		Ruptured..			3d day.	20/50	Some capsular remains;
11	70	L.	M.	190/140					14th d..	20/30	Delayed healing; vitreous protruded 3d day; seven punctures made; firm union.
12	75	R.	Im.	210/160					2d day.	20/70	Feeble minded; delusions; vision partly estimated.
13	58	R.	Im.	180/140			Slight..		3d day.	20/50	Incipient chorioiditis.
14	56	R.	Im.	145/110					5th d..	20/30	
15	67	L.	M.	180/130					3d day.	20/20	
16	60	R.	Hm.	190/130	Spatula..		Slight..		4th d..	20/70	Myopia and chorioiditis.
17	45	R.	M.	140/100					2d day.	20/30	
18	61	R.	M.	190					3d day.	20/20	
19	76	L.	M.	200/130					3d day.	20/20	
20	50	R.	Im.	136					5th d..	20/15	
21	72	L.	M.	115/75					4th d..	20/20	
22	76	L.	Im.	250/175					4th d..	20/40	Slight keratitis cleared up; incipient chorioido-retinitis.
23	71	R.	Im.	140/90					3d day.	20/20	
24	63	L.	M.	190/115			Slight..		12th d.	20/20	Delayed healing; no cause apparent.
25	62	L.	M.	170/130					5th d..	20/20	
26	62	R.	Im.	165/130					5th d..	20/20	
27	65	R.	M.	180/130			Slight..		3d day.	20/20	Small hemorrhage into anterior chamber.
28	75	L.	M.						2d day.	20/20	
29	64	L.	M.	145/105					4th d..	20/20	
30	70	R.	M.	150/110					2d day.	20/20	
31	62	L.	M.	150/110					4th d..	20/20	
32	68	L.	M.	130/90		Ruptured, retained.		Iritis.	3d day.	20/50	Oct. 20, '08, iridotomy; Jan. 29, '09, iridotomy.

33	64	L.	M.	160/120			Considerable..	Incarcerated..	14th d.	20/30	Pterygium, cystoid healing, abscised; healed.
34	87	R.	M.	180/140	Spatula..		Marked		3d day.	20/50	Feeble mentality; vision partly estimated.
35	77	R.	M.	140/90					3d day.	20/30	
36	77	L.	M.	230/150					3d day.	20/20	
37	70	R.	M.	170/130					4th d..	20/40	Feeble mentality (complained of pain during convalescence.)
38	70	L.	M.	170/130				Incarcerated..	14th d.	20/50	Feeble mentality; vision partly estimated.
39	65	R.	M.	160/110					4th d..	20/30	
40	68	L.	M.	140/110					4th d..	20/40	
41	76	L.	M.	150/100					2d day.	20/30	Delayed healing; a bead of vitreous abscised on fifth day. Prompt healing.
42	76	R.	Im.	150/100					5th d..	20/50	
43	77	R.	M.	160/110					4th d..	20/30	
44	73	L.	M.	220/160					3d day.	20/20	
45	73	R.	M.	220/160	Spatula..		Excessive..			P. L.	Restless; unruly; fluid vitreous.
46	77	R.	M.	230/150					4th d..	20/20	
47	87	L.	M.	180/140				Incarcerated..	17th d.	20/30	Feeble mentality.
48	64	R.	M.	160/120			Slight..		4th d..	20/30	
49	71	R.	M.	200/140					5th d..	20/20	
50	66	L.	M.	150/110			Slight..		7th d..	20/20	
51	75	R.	M.	150/110					4th d..	20/20	
52	73	L.	M.	160/120				Incarcerated..	19th d.	20/30	
53	69	L.	M.	150/120	Spatula..				3d day	20/20	
54	74	R.	M.	170/120					2d day.	20/20	
55	66	L.	M.	210/140					7th d..	20/20	
56	66	R.	Im.	210/140					2d day.	20/20	
57	86	R.	M.	170/130		Ruptured..			7th d..	20.50	Feeble-minded.
58	74	L.	M.	210/150					3d day.	20/50	
59	63	R.	M.	220/140					7th d..	20/20	
60	61	L.	Im.	185/110	Spatula..				7th d..	20/30	
61	67	L.	M.	160/120					14th d.	20/40	
62	72	R.	M.	190/140					3d day.	20/30	
63	72	L.	Im.	190/140					3d day.	20/30	
64	67	R.	M.	200/150	Spatula..		Slight..		3d day.	20/30	Slight keratitis cleared; very unruly
65	63	R.	M.	150/120					5th d..	20/30	
66	53	L.	Im.	140/100					10th day	20/15	Stripped keratitis. Cleared up.
67	81	R.	Im.						3d day.	20/70	
68	73	L.	Im.	200/160					4th d..	20/20	Slight keratitis cleared up.
69	67	R.	M.	150/120			Slight..		3d day.	20/20	Unruly; vitreous escape after delivery.
70	76	R.	M.	160/120					3d day.	20/40	
71	70	L.	Hm.	140/120					3d day.	20/70	Incipient macular chorioiditis.
72	74	R.							3d day.	20/50	
73	70	R.	M.						3d day.		
74	88	R.	M.								Infection; panophthalmitis (chronic cystitis.)
75	66	L.	M.	150/110			½		7th d..	P. L.	Very unruly; globe collapsed; salt solution injected; invagination corneal flap; probable hard detachment of retina.

Usually it is best to bandage the eye as soon as possible. Intrusion of fluid vitreous into the wound has not influenced the healing materially. The toilet should be made according to the taste of the operator. I prefer a half-round dry cotton patch between layers of gauze laid on the closed lids of each eye. The irregularities are filled out with small pledgets of picked cotton which are held in place by pieces of adhesive straps. I prefer a flannel bandage for the class of patients on whom most of my operations have been performed—old soldiers, many of them feeble in mind and body, bad subjects, as a rule, for any operation. The eye should not be open for three or four days unless the patient complains of pain, which is so exceedingly rare that freedom from discomfort or pain can be regarded as one of the striking characteristics of the method. On removing the dressing freedom from discharge and the normal appearance of the eyelid are usually noticeable. As a rule, the wound will be found closed and the chamber established at this first dressing; if not closed the usual conditions will be found present. The appearance of the clear black pupil is very gratifying and the distinct tracery and pattern of the iris, and its response to atropin, if used, shows its freedom from inflammation. The conjunctiva usually takes on a blush of irritation or congestion. Rarely will the deep redness and chemosis of inflammation be seen. The amount of photophobia and lachrymation will vary with the condition of the wound. I have seen only two cases of genuine iritis in these seventy-five cases. In each the capsule had ruptured and only two scissors operations have been made for up-drawn pupils in these same cases. Striped and latticed keratitis have caused more uneasiness and required more treatment than any other complication, yet, as already stated, I have not seen either remain and lower vision in any of these cases, and the same can be said of disturbance in the epithelial layer of the cornea from the pressure.

As a rule vision is not high at first, because of keratitis in some cases, but more often from disturbance in the anterior layers of the vitreous which resemble miniature ridges and valleys, running perpendicularly. These have not the appearance of striped keratitis. Pagenstecher noted this condition.[12] They seem to smooth down gradually and vision reaches its best in from one to two months. The degree of astigmatism, which is usually from 2 to 4 D., has not varied much with the healing, when primary union has taken place.

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[12] Ann. d'ocul., September-October, 1871.

For the purpose of comparing the results obtained with the combined method of extraction, I have appended the statistics of the last 75 operations in the Soldiers' Home Hospital, with the statistics of 75 cases in which operation was done by the Smith method; 52 of them in the Soldiers' Home Hospital and 23 in St. Elizabeth's Hospital. The conditions have been as uniform as possible. The average age of the patients operated on by the regular method was 69.76, and for the Smith operation 67.7 years. The number of patients operated on by each method is too small to draw any definite conclusions from, yet they are believed to have sufficient value to justify comparing them.

Expression within the capsule offers the following advantages:

1. A cataract can be removed at any stage.
2. No discission is ever necessary.
3. There is comparative freedom from postoperative inflammations.
4. There are no capsule entanglements; prompt healing is the rule.
5. The method is especially adapted to institutional work; one operation does all.
6. No ripening methods need be tried.
7. The result is better average vision, which does not change with time, if the fundus conditions remain favorable.

The disadvantages of the method are the following:

1. The only important one is greater liability to loss of vitreous for the average operator.
2. From a cosmetic point of view the wide updrawn pupil (if it results) mars the appearance of the eye, while it may not be a disadvantage to vision.
3. And lastly, a skilled assistant is always necessary in performing the operation.

It is not my desire to be considered a champion of the Smith operation until I have seen more of the ultimate results which follow it, among white people, living under different climatic and dietetic conditions than average Indians, but having emphasized its strong points, and admitted its weak points as I have seen them, I can not avoid the conclusion that it is a long step toward the ideal operation which we are all seeking. Perhaps it will not be so in the hands of every one, but in the hands of the few who will learn to do it well I believe it has a promising future. The statistics which follow have been compiled by my hospital associate, Dr. J. W. Millette. It is a pleasure to acknowledge the thorough and careful manner with which it has been done.

These statistics do not include all the operations I have performed. Cases have not been selected, but I have excluded all cases in which I have not given the after-treatment and done the testing for vision and also those cases which from the condition of the cornea, vitreous and fundus, were not proper ones from a visual point of view. Two patients whose cases were considered proper had excessive loss of vitreous and vision only equal to P.L., which are fairly chargeable to the operation; one has dense vitreous opacities, probably from hemorrhage, the other lost about half of the vitreous. He has an up-drawn pupil, and no view of the fundus can be had. I am not certain as to the cause of the loss of vision in either case, and one other case was lost from infection.

SUMMARY OF VISUAL RESULTS

Total Number.

Cases.	Vision.
3	20/15
30	20/20
18	20/30
4	20/40
9	20/50
5	20/70
2	P. L.
1	0
3	No record
75	

With Loss of Vitreous.

Cases.	Vision.
4	20/20
2	20/30
3	20/50
2	20/70
2	P. L.
13	

With Rupture of Capsule.

Cases.	Vision.
1	20/20
3	20/50
4	

Average vision for 72 better than 20/30 about 20/27, to be exact.

SUMMARY OF ACCIDENTS AND BLOOD PRESSURE

Vitreous loss; healing delayed more than five days.

Cases.	B. P.
3	150
2	160
3	180
3	190
1	200
1	220
13	Average B. P. 175.4

Cases.	B. P.
2	150
2	160
2	170
1	180
1	185
3	190
2	210
1	230
14	Average B. P. 174.6

For comparison, with results obtained by the Smith operation, I submit the following statistics from my last seventy-five regular operations compiled by my assistant, Dr. W. C. Cook, National Military Home:

Cases.	Vision.
10	20/20
16	20/30
17	20/40
10	20/50
5	20/60
2	20/70
7	20/100
5	20/200
1	Fingers at 6 feet
1	P. L.
1	0
75	Average vision 20/40

One patient had slight loss of vitreous; 2 had slight attacks of glaucoma; 20 had a degree of iritis requiring treatment; 35 eyes required discission; 9 patients whose eyes needed discission refused it; 1 eye was lost by iridopyelitis after a secondary operation.

MEMORANDA

EPIBULBAR LEUCOSARCOMA

EXENTERATION OF ORBIT. NO RECURRENCE AT ORIGINAL SITE. DEATH IN THREE MONTHS FROM METASTASES*

CASEY A. WOOD, M.D.
CHICAGO

New growths on the eyeball, whether malignant or other, are so rare that a detailed history of an example of them seems worth while. I have been able to follow the course of a neoplasm of this kind almost from its beginning to its termination, and I take the liberty of reporting it.

Patient.—Miss J. O., aged 15, was referred to me Sept. 9, 1908, by Dr. F. W. Kettlestrings and Dr. H. S. Warren of Chicago, on account of a swelling of the right eyeball.

History.—She had had no trouble with her eyes except a slight discharge from them when a few days old, but this infection had lasted only a short time and had left no trace. From that time there was nothing particularly noticeable about her eyes until May, 1908, when the right lid became swollen. This was followed by an extension of the swelling to the eyebrow, after which it was noticed that the upper lid began to droop. Four weeks later the condition became decidedly worse and was shortly followed by pain in the eyeball itself. At that time she also noticed that any attempt to use the eye longer than half an hour—as in reading or studying—was followed by decided fatigue. Dr. Warren reported that the general health had been only fair, although the blood and urine, as well as the internal organs, were practically normal. She had been taking a simple iron and strychnia tonic. Her family history is negative so far as the occurrence of tumors is concerned and the patient has had none but the usual (infantile) complaints.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

Examination.—On examination of her nose by Dr. Frank Brawley the patient gave a history of a yellowish-green discharge from the left nostril, but this had stopped before the eye trouble set in. She showed normal nares except a left posterior hypertrophy of the inferior turbinal. Coming to a nearer examination of the eyes vision of the left eye was normal; the patient read 6/5 minus and Jaeger 1, with good range. The sight of the left eye was 6/9 minus; Jaeger 1, not so well as on the right side. The fundi in both eyes were entirely normal. There was an apparent protrusion of the right eye. On raising the upper lid, which was distinctly swollen and covered about one-half the pupil, there was exposed a large, tense, semitransparent swelling which involved about one-half of the upper aspect of the globe. On digital examination, the tumor gave an elastic sensation and showed that it was in all probability confined to the superior bulbar surface and did not extend posteriorly. It was slightly tender to the touch, but the conjunctiva covering it was entirely smooth and non-adherent to the growth beneath. There was no true proptosis, although the globe and lids seemed more prominent than on the opposite side. The excursions of the eyeball were as extensive in the affected eye as in the normal eye. There was no evidence of changes in the cornea or in the sclerotic or overlying conjunctiva for 5 mm. around the sclerocorneal junction, and on using the ophthalmoscope there seemed to be, so far as it was possible to see, no involvement of the anterior segment of the internal eye. The new growth was apparently quite circumscribed, attached to the sclerotic but did not to any appreciable degree involve any portion of the conjunctiva. The right preauricular gland was rather hard, well-defined and slightly prominent. The corresponding submaxillary and anterior cervical glands were more easily outlined on the right side than on the left.

Roentgen Examination.—An *x*-ray picture of the orbital contents and neighboring cavities showed that there was no bone enlargement and probably no extension of the tumor mass behind the eyeball; indeed, the ocular swelling seemed during the whole time of observation to be confined to the upper half of the globe and to diminish in volume below the equator. A number of further observations were made of the neoplasm. Dr. Reichmann made several skiagrams and believed that the *x*-rays indicated an orbit on the right side freer of skiagram shadows than the left orbit.

Diagnosis.—The Calmette test gave a negative result in the opposite eye. That the patient might have the further benefit of any doubt, very large doses of iodid of potassium were given, in conjunction with pilocarpin sweats, for two weeks, with but slight benefit, except perhaps some reduction in the prominence of the preauricular gland. Under these circumstances and since the tumor showed a smooth surface, appeared to be circumscribed, was of elastic consistency, whitish in color and was associated with a preauricular enlargement, a diagnosis was made of probable subconjunctival leucosarcoma arising from the tissues about the superior rectus insertion.

Microscopic Examination.—To settle the question, under a general anesthetic a section of the tumor was removed on Oct. 1, 1908, and examined by Dr. Dagg of the Chicago Laboratory. His report is as follows:

"The tissue has the appearance of a small round-celled sarcoma. There are no well-formed vessels. One margin of the section shows diapedesis of red cells into the tumor mass. Mitosis of the cells not noticed."

The wound of operation healed kindly in a few days.

Although the right preauricular gland seemed to be less swollen after the treatment just outlined the group of glands at the angle of the jaw shortly became more prominent. In consultation with Dr. A. E. Halsted and after considering the *pros and cons* of the case, it was decided to do a combined exenteration of the orbit, with removal of the enlarged preauricular, submaxillary and cervical glands; in fact, of all the grandular tissue that seemed to be affected by the disease. On October 8, with the assistance of Dr. Halsted, this operation was successfully carried out.

Postoperative History.—On October 20 the skin incision involved in the removal of the extraocular glands had closed nicely and the patient did not complain of pain either in it or in the exenterated orbit.

Oct. 29, 1908: The patient left the hospital for her home. For some weeks after the orbital exenteration the Roentgen rays were used, as a remedial measure, within the orbital walls.

Dec. 29, 1908: The patient was able to go out of doors and was apparently doing well. The orbital wound showed no recurrent deposits and the external wound of operation had entirely healed.

Pathologic Report on Tumor.—From the pahtological department of St. Luke's Hospital I obtained the following report on the removed preauricular and submaxillary glands:

"The mucous glands from both sides show infiltrations of round cells about the ducts, corresponding to the small round cells of the primary tumor of the orbit, and are, therefore, to be regarded as metastatic small round-cell sarcomata."

I am indebted to Dr. Francis Lane for a report on the orbital mass which, of course, includes the eyeball, most of the conjunctiva and other tissues of the orbit.

"Gross Findings: Right eye; bulb collapsed with furrow horizontally across the cornea (globe injected with formalin 4 per cent.); mass of orbital fat surrounds a 9 mm. long optic nerve and extends forward to superior rectus; superior rectus severed 26 mm. back of corneal margin with cut surface, oval, 7x14 mm.; circular cut of conjunctiva varying from 9 to 11 mm. from limbus; upper inner quadrant of conjunctiva brownish color, wrinkled and folded, apparently much thickened and immovable over underlying sclera; bordering upper one-half of cornea the conjunctiva *bombé*-like and slightly overlaps superior quadrant of corneal margin; an irregularly shaped central defect of corneal epithelium. Deep in cornea are number of zigzag, for most part horizontally directed, narrow striæ [posterior to horizontal corneal groove and due to collapse (?)]; superficial, uniform, moderate corneal haze somewhat obscures an apparently normal anterior chamber and anterior iris surface; pupil round, 3.5 mm. diameter; lens opaque [postmortem (?)].

"Coronal Section: Anterior segment—sclera thinned; upper one-third of cut surface shows sclera covered with a closely adherent 0.5 mm. thick, firm, pinkish layer, from which capsule and conjunctiva can be easily stripped, leaving behind very delicate strands of connective tissue [Tenon's space (?)]. Lens yellowish-white and slightly translucent with transmitted light. Vitreous streaked and filled with innumerable very fine highly refractile particles (not crystals, but globules as shown under microscope). Posterior segment: retina very white color, cloudy vitreous hides details of disc head; inner lower half of sheaths of optic nerve thickened to 3 mm. and movable over nerve trunk.

"Anatomical Diagnosis: A small, round-celled sarcoma. The tumor cells always appear to surround the blood vessel walls or to lie in the perivascular lymph spaces and are never found within the lumen of the blood vessels themselves."

Remarks.—The last time I saw the patient she seemed very well. It was evident there was no recurrence of the neoplasm either in the orbit or in the neck or lower jaw. However, in March, 1909, the family physician reported to me that although she had had several attacks of *malaise* of apparently trivial import, she was quite well until the end of January when, after some obscure symptoms referable to her abdomen, she died rather suddenly of what Dr. Warren believes to be an abdominal metastasis, without the slightest appearance of the tumor in its original locality. No autopsy.

Anyone examining the bibliography of epibulbar tumors will find that, although the sarcomata are probably the commonest of the malignant forms, they are not so common as one might at first imagine. LaGrange[1] gives full references to all of these new growths described to the year 1900.

72 Madison Street.

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[1] Traité des Tumeurs de l'Oeil, vol. I, pp. 237 to 246.

MEMORANDA

MEMORANDA

RODENT ULCER OF THE CORNEA (MOOREN'S ULCER) *

ROBERT L. RANDOLPH, M.D.
BALTIMORE

It is a matter of some surprise to me to find how few reports of cases of so-called rodent ulcer of the cornea have found their way into literature; according to Hayashi not more than thrity-five. I can not be think that this is due in many instances to the fact that the condition has not been recognized as rodent ulcer but was regarded perhaps as a marginal ulcer of the serpiginous variety and treated accordingly. That most ophthalmologists whose experience extends over a number of years have met this variety of corneal ulcer once at least is very likely and that it has not been identified as the rodent ulcer first described by Mooren is equally probable. I say first described, for it was really Mooren whose name is most closely associated with the condition, though reference to the same condition will be found in the writings of Bowman many years previously.

At first sight this form of corneal ulcer does not differ from that form which every ophthalmologist sees now and again, and which he recognizes as a marginal ulcer. Indeed I doubt whether it is possible to identify clinically a rodent ulcer in its early stages, and this uncertainty will prevail until we learn more of its etiology. If it be due to a specific bacterium (and I think it is) it would be possible to identify it at once, but from a bacteriologic point of view no conclusive light has been thrown on this variety of corneal inflammation, and we are confronted with a disease which in many respects is similar to what we now and again see in hospital

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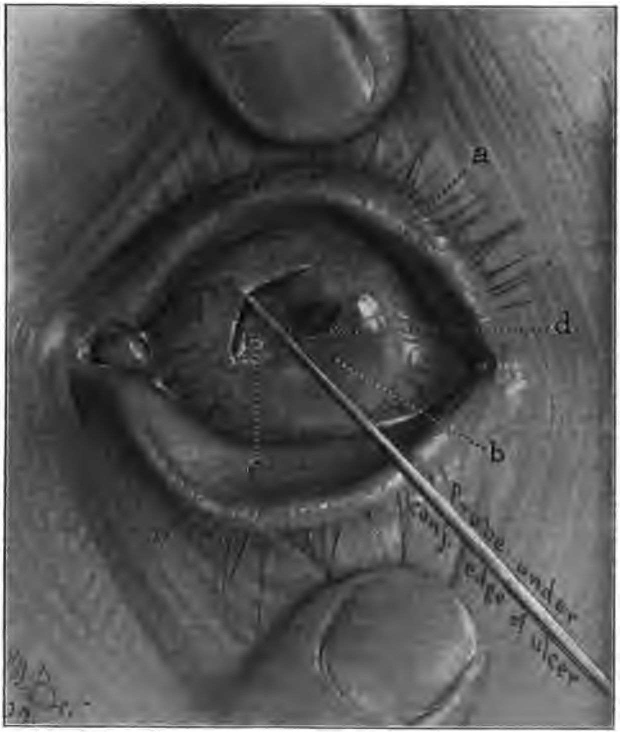
practice. Even after the disease has been present for many weeks, it is more than probable that most of us would fail to realize that we were dealing with anything more than a very obstinate corneal ulcer. And yet there was in the only case which I have seen one feature at least which distinguished it from any other ulcer of the cornea which I had ever seen, and that was the deeply undermined conjunctival edge of the ulcer. The edges of marginal ulcers are always more or less undermined, but owing to the better blood supply at the conjunctival edge this edge of the ulcer is not conspicuously undermined, except probably in the case of rodent ulcer.

In the case reported here the conjunctival edge of the ulcer could be lifted up and was undermined to the extent of at least four or five millimeters from the border of the cornea. This, I believe, is peculiarly true of the rodent ulcer and it will be found in the early stages of the affection. It is well known that the corneal or progressive edge of the ulcer is always undermined to the extent of two or three millimeters, but I do not think that attention has been sufficiently called to the extensive invasion which goes on beneath the limbus, and which is often lost sight of. My attention was called to this feature in about three weeks after the patient came under my care. McNabb and others mention the possibility of the process extending under the limbus, but it seems to me that careful examinations of the conjunctival border of the ulcer should be repeatedly made, for we can hope to mitigate this condition only by addressing our remedies to those parts of the ulcer where undermining is in active progress. In my case the corneal edge was distinctly undermined and could readily be elevated and a probe passed into a cleft two millimeters in extent, while, as I have said, the conjunctival edge was undermined to twice the extent of the corneal edge.

Chronicity is an important point in the history of rodent ulcer. The average length of time is from two to ten months and occasionally the condition lasts for more than a year.

History.—The patient (E. T. L.) whose case I report was under treatment since early in July, 1908, two months before he came to me. He was and still is a healthy looking man of 52 and has a well-marked arcus senilis on his right eye. The ulcer of his left cornea obliterated the arcus on that eye. He had never had any serious sickness. He gave a rather vague history of an injury to his left eye early in June. About this time he had a foreign body removed from his left eye by an optician and from that time his troubles with the eye commenced. He first consulted an ophthalmologist in July. All this time, however, he continued his occupation, which was that of a traveling salesman in connection with a big business concern



Rodent ulcer of cornea. a. The only part of the cornea which has not been attacked but which is gradually becoming hazy with approach of the ulcer. b. Area of cicatrization after ulcer has run its course. c. Ulcerative process is especially marked in this area (note undermined limbus). d. Corneal or progressive edge of ulcer, showing crescentic shape which was more marked in early history of case. This edge is also undermined.

in Baltimore. In July he returned from a trip south and, according to the oculist who then saw him, he had a marginal ulcer which occupied the outer and lower fourth of the left cornea. When I saw him in September this was still his condition, though his attending physician said that the ulcer had spread considerably since he first saw him.

Treatment and Course of Disease.—I found that the patient had been using massage with yellow ointment and irrigations of weak sublimate solution. I advised the application of pure carbolic acid, the instillation of atropin preceded by an irrigation with normal salt solution and also that he be put on tonic treatment, quinin and strychnin. This was done and with apparently no benefit, for he returned in ten days with a request from his oculist that I take charge of the case. There was then the remarkable undermining of the limbus which had escaped me at the patient's first visit ten days previously and which no doubt had been there all the time. This undermining is well shown in the accompanying drawing. I felt sure now from the superficial character of the involvement, the undermined edges, and the fact of its having been present for over three months that I was dealing with a rodent ulcer. The galvanocautery was applied and particular care was taken to pass the loop well up under the limbus and under the progressive edge of the ulcer as well. Fluorescin proved a help in bringing out the areas which were affected. There was marked improvement right away, and in three or four days new blood vessels were seen making their way over the affected part from the conjunctiva. The atropin and internal medication were of course kept up. Ten days later the eye became intensely red, the irritative symptoms broke out afresh, and several new spots appeared at the upper end of the ulcer. These spots in a few days coalesced and fused into the main ulcer. Again the cautery was applied as before and again a few days later to another point which I thought I had overlooked. Apparently this last application was of distinct benefit, for improvement continued up to the middle of October, when he had another outbreak, and I determined to try tincture of iodin. Three applications were made in one week, but there was not the slightest benefit obtained, contrary to what is usually obtained when we use iodin in the ordinary forms of marginal ulcers of the cornea. Iodin is my main reliance in corneal ulcers in this situation. Between November 1 and Christmas two more applications of the galvanocautery were made and always with improvement, but this lasted for only a few days. At the last application improvement was so pronounced that I promised the patient his Christmas dinner at home. Much of the redness of the eyeball had disappeared and much of the cornea was clear. The patient left the hospital to return home, where he spent his Christmas with a black patch over the eye. The next day the process started up with unusual vigor and I concluded that the galvanocautery had been given a fair trial. About this time I picked up a communication by Risley, who had seen benefit follow the administration of thyroid extract in obscure cases of keratitis. The patient took three grains of this three times daily, but in a week had to leave it off, as it produced diarrhea even when smaller doses were given. This is a not infrequent occurrence during the administration of the so-called thyroid treatment. I then tried the application of trichloracetic acid, and this, I am sure, made the condition worse. Only one application of this was made. It was then suggested that I try a light compress bandage and a boric acid ointment between the lids. This was faithfully tested for ten days but without any benefit. A great deal of the time in the early history of the case I had tried the instillation of chlorin water several times daily, but I could never see that it did the slightest good. The last measure tried was recourse to tincture of iodin and a light immobilizing bandage again, but the result of all this was the infliction of a great deal of suffering without the slightest improvement. The iodin was applied six times over a period of two weeks. What seemed peculiar was the fact that none of the many remedies employed seemed to accelerate or to retard the process, which went on slowly and surely to take in the greater part of the cornea. I might except trichloracetic acid, which probably did harm. Soon after Christmas a very light grade of plastic iritis developed, which was controlled. There have been times when the ciliary enuralgia has been intense, but the patient has slept and eaten well and, generally speaking, has been comfortable. The only measure which seemed to promise recovery was the galvanocautery, and more than once I thought the chances fair, but no substantial, certainly no permanent advantage over the process was gained, and I would return to the patient's room the next day to find his eye in a blooming state of inflammation. It seems strange that Fuchs should give such a favorable prognosis in this class of cases if the galvanocautery is used, when in by far the majority of cases reported the cornea was ultimately covered by a leucoma in spite of this treatment in the hands of skilful observors. At the present writing the patient's cornea is gray all over, with the characteristic anesthesia of most of the surface and he counts fingers at one foot.

It may be interesting to note the following data in reference to the frequency of rodent ulcer. Hayashi saw rodent ulcer 4 times in 11,400 eye cases. Mooren in twenty-five years saw the affection 20 times in 108,417 cases; Jany once in 66,000 cases; Vossius 3 times in 25,000 cases; Uhthoff 3 times in 10,000 cases; Hillemann failed to see the disease in 11,000 cases in the eye clinic in Bonn, but observed it 3 times in 7,000 cases in Utrecht. Asayama met with rodent ulcer 4 times among 6,780 eye cases; Kiribuchi saw it 6 times in 11,000 cases and Ogiu saw it 6 times in 8,900 cases. The disease would seem to be somewhat more frequent in Japan.

It is a mistake to think that the affection is a painless one, for it is characterized by frequent attacks of suffering and irritative symptoms generally. The ulcer shows a decided tendency to keep close to and run parallel to the limbus of the cornea and one of its peculiarities is its tendency to clear up and look as though it would be completely well in a few days, when a fresh outbreak would land the patient back at a point which was thought to have been left far behind days ago. The progressive edge of the ulcer at this times as Nettleship describes it, "forms a narrow sinuous whitish band level with the cornea of which the two ends are in advance of the centre, so that the diseased surface at this stage is roughly crescentic and sometimes semilunar." Behind this edge of course there is no epithelial covering and here the fluorescin stain takes well, and further back comes that part where the process has spent itself and the cornea has been left scarred, and it takes poorly the fluorescin stain. In most cases the disease dies out after the entire cornea has been involved, but according to McNabb recurrences take place in exceptional instances and the ulceration reappears to penetrate deeply into the cornea and expose Descemet's, membrane, or even to penetrate through it.

Rodent ulcer as a rule is seen in individuals over forty, though Pflüger, Handmann and Grossmann report cases in younger patients. It has probably never been seen in children. Men are more prone to it than women. Of the 36 cases, reports of which are found in literature, 25 were in men and 11 were in women. It is not infrequently found in both eyes. Hillemann, who has made an analysis of the subject, found that it occurred eleven times in both eyes, though I do not believe it has ever been observed in both eyes at the same time.

We are completely in the dark as to its etiology. There are those who maintain that disturbances in the sensitive nerve endings are largely responsible. Among these are Ahlström, who noticed anesthesia of the cornea in its entire extent, and Vossius, who in three of his cases observed somewhat similar phenomena. On the other hand there are others who have failed to observe this condition. I have thought at times that there was some anesthesia, but I could never get any convincing evidence of this condition except in those parts in which the nerve endings had plainly been destroyed. Ahlström holds that it is a kind of progressive necrosis of the superficial subepithelial corneal layers and of nerve origin. Kiribuchi touches on its probable systemic origin as evidenced by its appearance not infrequently in both eyes. Asayama found in four cases evidences in the stools of intestinal parasites, and thinks that there may be some causal connection. This possibility was excluded in my case. A study of the published cases throws no satisfactory light on the etiology of this rare and intractable disease, neither does it offer any remedy which carries with it more than doubtful value. I am inclined to the view that the disease is of bacterial origin.

I have almost made up my mind that my patient has done better since all aggressive treatment has been abandoned, especially in the way of irritating applications. If the galvanocautery had been applied a month or six weeks earlier I believe that the story would have been different. When one meets a corneal ulcer along the margin, careful examination should be made to ascertain whether the limbus edge of the ulcer is deeply undermined. If so the cautery should be applied at once, and it might be well to cut away with a delicate pair of scissors the overhanging edges of the ulcer so that parts underneath can be readily reached. If the disease shows no substantial improvement at the end of two months, I think it best to stop all irritating applications and simply continue with atropin and salt solution irrigations and tonic treatment. I am not at all satisfied that repeated applications of the galvanocautery are advisable, such applications often being followed by a denser opacity than is produced by the actual ulceration. McNabb states that some of these patients are left with a vision of from 1/6th to 1/4th.

816 Park Avenue.

MEMORANDA

THE MANAGEMENT OF ACUTE HEMORRHAGIC GLAUCOMA IN THE PRESENCE OF ADVANCED ARTERIOSCLEROSIS

CHARLES STEDMAN BULL, A.M., M.D.
NEW YORK

DEFINITION OF HEMORRHAGIC GLAUCOMA

A glance through recent ophthalmic literature would seem to show that there is some difference of opinion as to what hemorrhagic glaucoma is. It therefore appears proper to preface these remarks by a definition of the term "hemorrhagic glaucoma," as I understand it, as follows:

This disease presents the usual symptom-complex of acute glaucoma, occurring suddenly in an eye in which retinal or other intraocular hemorrhages have previously occurred before any glaucomatous symptoms were present. In brief, the hemorrhages are the exciting cause. I believe that this condition should be differentiated from a pre-existing glaucoma in which hemorrhages have occurred subsequently. Clinically, an operation might be indicated in the latter class of cases, but would not only be valueless but positively injurious in the former class.

The predisposing cause in every case is probably general arteriosclerosis. Hemorrhagic glaucoma is always caused by the intraocular hemorrhages, but these should be looked on as a local expression of disease of the general vascular system.

Most of the cases occur between the ages of 50 and 70, and the constant accompanying condition is general arteriosclerosis. Fundamentally, the primary reason for

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the outbreak is the loss of balance between the general blood pressure and that of the eye, and this leads naturally to a consideration of the subject of increased blood pressure, or, more correctly, of chronic arterial hypertension. In any attempt to define the relations existing between arteriosclerosis and arterial hypertension it should not be forgotten that there is no exact correspondence between the pathologic changes in the vessels and the clinical phenomena. It should also be remembered that the blood pressure is greater when the blood stream is slow, and hence the rapidity of the blood current is a factor to be considered in these cases.

DISTINCTION BETWEEN ARTERIOSCLEROSIS AND ARTERIAL HYPERTENSION

We should draw an absolute distinction between arteriosclerosis and arterial hypertension, though we may recognize the relation of cause and effect. The arteriosclerosis encountered in cases of hemorrhagic glaucoma is of mechanical origin, the result of long-persistent high blood pressure, and presents an actual pathologic alteration of tissue in the vascular walls. On the other hand, arterial hypertension is a cardiovascular condition, the perverted action and abnormalities of which we are now able to recognize; and by the correction of these perverted activities it may in the future be possible by the regulation of the habits of life, by diet and by proper administration of drugs, to prevent or limit the development of arterial degeneration. The earlier this condition of chronic arterial hypertension is recognized and its importance properly estimated, the better we shall be able to correct its destructive tendencies, for the functional perversion of vascular tension is amenable to treatment, and thus the real pathologic condition known as arteriosclerosis may be prevented.

If by giving a vasodilator drug we relieve some of the unpleasant or dangerous symptoms in a case of arteriosclerosis, we effect no permanent benefit in the diseased condition of the arteries, but simply correct the hypertension. How much more valuable would this treatment be if undertaken before the arterial degeneration had begun: We are now able to estimate accurately the degree of hypertension, and can follow it up and regulate its progress and tendencies; and before long we may be able to state that the advance of old age is in inverse proportion to the pulse tension.

This relationship of arterial hypertension and arteriosclerosis is a complex problem, which is by no means easy of solution. In some of the smaller arteries the arteriosclerosis seems to cause an increase in the tension through increased peripheral resistance. Another apparently contradictory fact mentioned by Broadbent is that patients with very marked hypertension do not generally live long enough to develop a high degree of arteriosclerosis.

As regards the etiology of this hypertension, it would seem that heredity is a considerable factor, especially in males. Mental strain is very influential in developing hypertension, as is constantly seen in patients subjected to continual care and anxiety. The pathologic changes noted in connection with this condition are cardiac hypertrophy and dilatation, hyperplasia of the tunica media, and later the arteriosclerotic kidney and advanced general arteriosclerosis with enfeebled cardiac action.

In endeavoring to understand the real pathology of this complex condition we must differentiate between arterial contraction and the blood pressure. We must be able to decide whether the thickening of the arterial coats is an apparent thickening, due to hypertension, or whether the change is real and due to actual arteriosclerosis. The hypertonus is not confined to middle and later life, but is met not infrequently in early life. Here it may produce no symptoms of which the patient is aware, because of the greater elasticity and endurance of youth. Russell has well said that even in vigorous persons with sound cardiac action, hypertonus leads at once to heightened blood pressure and increased arterial tension. But hypertonus occurs in people with enfeebled heart action, and to this class belong the elderly patients with hemorrhagic glaucoma. In the former class any temporary excitement causes increased cardiac action and increased blood pressure, but as soon as the one subsides the other disappears. In the latter class the thickened arteries are associated with a low blood pressure, for these patients have no margin of reserve force in their hearts.

TREATMENT

With this brief description of the two differing conditions of arterial hypertension and arteriosclerosis, and the probable relationship of cause and effect existing between them, I ask attention to the subject of the paper: How shall we best manage these cases of hemorrhagic glaucoma, associated with advanced arteriosclerosis?

As before stated, these patients are between 50 and 70 years of age. The high blood pressure, with the increased vascular tension found in these patients, tends to raise the intraocular tension beyond the normal limits and well within the danger zone, and then the slightest exciting cause may bring about a retinal or vitreous hemorrhage, which in its turn precipitates an attack of glaucoma. If the patient is hypermetropic, with shallow anterior chamber, the more or less complete obstruction of the channels of filtration is almost certain to occur, ending in the explosion of acute glaucoma. Now the classical operation for acute glaucoma is iridectomy, but we all know what has been the well-nigh universal experience of the results of iridectomy in hemorrhagic glaucoma. The sudden lowering of the intraocular tension almost invariably causes a sudden profuse intraocular hemorrhage, either subretinal or into the vitreous, which again increases the tension above the normal, the return of the severe pain, which can not be controlled, and ultimately necessitates the removal of the eyeball. Moreover, the operation of iridectomy on an inflamed eye is excessively painful and can not be done under cocain anesthesia, but demands general anesthesia. We are thus brought face to face with the actual dominating feature of the situation. These patients with extensive cardiovascular changes, with cardiac hypertrophy, enfeebled heart action and brittle arterioles, are not fit subjects for general anesthesia, because of the danger of a fatal termination. I believe that no operation, whether iridectomy or sclerotomy, which requires general anesthesia should ever be done in a case of hemorrhagic glaucoma in which the extensive cardiovascular disease makes the use of general anesthesia dangerous to the life of the patient. We have other means at our disposal for allaying the pain, reducing the increased tension and arresting the glaucomatous process, even though the eye remains permanently blind.

The immediate treatment for the relief of the urgent symptoms is the same for all these cases, but the subsequent management must vary with the age and general condition of the patient and his environment.

The immediate local treatment which I employ in these cases of hemorrhagic glaucoma, in order to avoid the dangers of an iridectomy, is as follows: Under the influence of a 10 per cent. solution of cocain, instilled as often as may be necessary, a careful paracentesis of the cornea is done, the instrument being withdrawn very slowly, and thus the aqueous humor is allowed to flow out only drop by drop from the anterior chamber. In this manner the intraocular tension is lowered very slowly, and the danger of the occurrence of an intraocular hemorrhage is reduced to a minimum. Two or three leeches are then applied to the temple and allowed to fill themselves until they drop, or the Heurteloup wet cup may be used, and the bleeding kept up as long as may be necessary. Then a solution of eserin sulphate, one grain, and pilocarpin hydrochlorate, four grains to the ounce is instilled every hour, until under the combined influence of the paracentesis, the local bleeding, and the miotics, the eye softens and the pain grows less. As the intraocular tension subsides and the intravascular tension diminishes the pain grows less and the instillation of the miotics may be less frequent. Then the patient is directed to have hot applications made to the eye for half an hour at a time, and to repeat them every two hours until the pain has gone. He is also directed to take twenty drops of the fluid extract of Jaborandi three times a day, as a valuable additional means for lowering the intravascular tension. Hypodermic injections of pilocarpin will bring about the same result, but in the presence of advanced cardiac disease they are dangerous. It is sometimes necessary to repeat the paracentesis of the cornea several times in the same careful way at varying intervals, but eventually the end is accomplished.

As the symptoms subside the use of the miotics may be reduced to two or three times a day, and then the all-important subsequent management of the patient begins. This must vary with the age and general conditions of the patient and the severity of the type of lesion. The habits of life must be regulated, being modified in strictness by the patient's general condition and environment. It must be largely dietetic, based on accurate knowledge of the patient's digestive capabilities. The administration of drugs, except to combat certain symptoms as they arise, is of secondary importance. The daily life of the patient should be rendered as quiet as possible, and there should be great moderation in eating and drinking. As an abundant meat diet raises the blood pressure, the amount of meat eaten should be strictly limited. For some of the patients a certain definite amount of systematic exercise, preferably walking in the open air, should be insisted on, because it increases oxidation, improves the capillary circulation and aids in the process of elimination. If the patient has been addicted to the use of alcohol in moderation, it is not wise to interdict it, but it should be taken largely diluted, for while dilating the peripheral blood vessels, it does not increase the blood pressure to any appreciable extent. Tobacco should be forbidden or only slightly indulged in, because it contracts the blood vessels and raises the blood pressure. Water should be drunk freely between meals in order to aid elimination. The products of faulty metabolism which induce hypertension only exercise their toxic effects while in the circulation. The uric acid group are largely eliminated through the kidneys, and the best renal eliminants are the salts of soda, potash and lithia. Each patient should be carefully studied for himself, and the dietetic and therapeutic management varied according to the necessities of the case. In many of these patients the arteriosclerosis has forced the heart to do an excessive amount of work, and at the slightest additional burden the heart may collapse in spite of the administration of a cardiac stimulant. If the general symptoms are severe the first necessity is rest in bed, and this must be enforced if we are to expect any improvement. Muscular exercise often produces temporarily a rise in vascular tension, and complete muscular rest in bed removes the additional strain and gives the heart a chance to recover its tone. I believe that in many, if not in all, these chronic cases in high-strung nervous individuals, the cardiovascular sedatives ordinarily employed are almost useless unless rest of the body in bed is insisted on.

To reduce the high blood pressure I employ sodium nitrite as a vasodilator, but always in combination with some reliable heart tonic. It is a wise precaution to have a thorough examination made of the heart and general circulation before prescribing any cardiac drugs, especially the vasodilator drugs. A rapid, irregular or intermittent pulse, with a rapid pulse rhythm, and with a systolic murmur, probably indicate a cardiac inadequacy with high blood pressure, and in such a condition cardiac stimulants should be combined with vasodilator drugs. A high blood pressure does not necessarily mean the need of the prompt administration of drugs, for Nature often maintains a cardiac integrity, in spite of the circulatory resistance, and the apparently urgent symptoms yield gradually without the use of drugs to reduce the pressure. If the blood pressure be over 200 millimeters of mercury, the vasodilator should be slowly administered and its effect on the blood pressure, the pulse rate, and the sensations of the patient carefully noted. The subjective sensations of the patient are often a better means of judging of the effect produced than the blood pressure or the pulse rate. The administration of a full dose at once might cause very serious consequences to the patient's heart.

I have tried the systematic employment of hot baths, as recommended by Hirschfeld, but with indifferent success. The hot bath changes the distribution of the blood pressure by unloading the internal organs and increasing the vascularity of the skin, and should afford prompt relief from pain. It also increases the elimination of waste products. But we must note very carefully the reaction of the patient, and it is not safe to begin with a temperature of more than 100 degrees. The duration of the bath should not be over ten minutes. Of course, the deciding factor in the application of the hot bath should be the condition of the heart and the quality and distinctness of the first and second aortic sounds. Without a fairly vigorous heart the hot bath must be very cautiously employed.

Following the suggestions of Erlenmeyer and Fraenkel, I aways give the patients some preparation containing iodin to reduce the viscosity of the blood. Either potassium iodid or sodium iodid, by diminishing the frictional resistance of the blood current, causes a diminution of the obstruction to the circulation and brings about better nutritive conditions in the parts. I give the potassium iodid in progressively increasing doses, changing to sodium iodid when the stomach revolts, and continue the use of the one or the other drug for a period of weeks or even months. When the administration of these drugs must be suspended for a period I advise the patient to take in the interval two or three drops of a 1 per cent. solution of nitroglycerin.

47 West Thirty-sixth Street.

MEMORANDA

MEMORANDA

PRIMARY HEMORRHAGIC GLAUCOMA, WITH PROBABLE SYMPATHETIC INFLAMMATION

A CLINICAL AND MICROSCOPIC STUDY OF A CASE *

MORTIMER FRANK, B.S., M.D.
CHICAGO

Pathologic investigations on hemorrhagic glaucoma have been reported in considerable number, but, owing to the peculiar combination of conditions, this case is of interest and we can, I think, make some useful deductions from it. It points out that a broad iridectomy freeing the filtration angle is not always a preventive of glaucoma, although iridectomy is the best method of treating acute glaucoma. It further indicates that diminished depth of the anterior chamber and narrowing or closure of the filtration angle are not absolutely essential for the production of glaucomatous symptoms, but that, perhaps, hypersecretion and changes in the character of the fluids of the eye impeding proper filtration are sometimes concerned in the onset of glaucoma.

History.—On the evening of Jan. 14, 1908, I was called to see M. R., a man aged 34, of Jewish extraction. The day before the patient went to Milwaukee on a business trip feeling as well as usual, never having had any severe illnesses or any previous eye trouble. Twenty-four hours prior to coming under observation he suffered with a sudden severe pain in the left eye and within two hours the globe was intensely injected and vision lowered so that he could barely make out his hand. After a long anxious night a well-known Milwaukee ophthalmologist was consulted in the morning, who told the patient to get back to Chicago as quickly as possible.

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Examination.—The left eye presented all the cardinal symptoms of acute glaucoma. The globe was brick-red, the scleral vessels full and the cornea steamy with diminished sensibility. The anterior chamber was equal in depth to its fellow and contained about two minims of dark-colored blood. The pupil was fully dilated and immobile, with the characteristic glaucoma reflex. The tension was plus 3. An ophthalmoscopic examination was impossible, owing to the corneal haziness and cloudiness of the vitreous. The vision was reduced to counting fingers at two feet. To the temporal side of the globe there was a large subconjunctival hemorrhage extending to the limbus and at places in the vicinity the cornea appeared to be studded with minute hemorrhages. The field of vision was about normal. The pain in and about the eye was very severe. The fellow eye was absolutely quiet and normal in every detail; vision equaled 6/6 plus. A careful inquiry into the patient's previous history failed to ascertain any cause for the attack. His habits were the best; he drank and smoked only very moderately. There was no venereal history. It was evident that one had to deal with a case of primary glaucoma of the hemorrhagic type. As iridectomy was not advisable, the usual medicine remedies, eserin (physostigmin), one grain to the ounce, combined with cocain, dionin (ethyl-morphin hydrochlorid) 5 per cent., leeches and hot fomentations had to be relied on.

January 15, 8:30 a. m.: The patient had a good night and was now free from pain; T + 3; cornea hazy; anterior chamber of normal depth; pupil widely dilated, but hyphema less marked.

January 15, 8:30 p. m.: Condition was the same, except that the blood in the anterior chamber has absorbed, and cornea clearing and sensitive. T + 3; vision equaled fingers at six feet. On careful repeated chemical analysis and microscopic examination the urine proved normal.

January 16, 8:30 a. m.: The pupil was still dilated, but tension slightly lower than the day before. Cornea was bright and vision equaled 3/60. Ophthalmoscopy was now attempted, but owing to the cloudiness of the vitreous, the details of the fundus were not sharply defined, although the disc could be seen and appeared hyperemic, swollen and the vessels full. The retina could not be studied.

January 16, 8:30 p. m.: Since noon the patient had had some pain; the cornea was again steamy and vision reduced to fingers at two feet. The field was good. Paracentesis of the cornea was advised and concurred in by Drs. Casey Wood and Thomas Faith. On account of the previous hyphema and the still present subconjunctival hemorrhage, iridectomy was advised against.

January 17, 8:30 a. m.: The patient's condition was the same. Under 10 per cent. cocain anesthesia, the anterior chamber was slowly emptied with a paracentesis needle, and immediately on the escape of the aqueous small rivulets of blood made their appearance on the iris. Tension was slightly lower than normal, and iris somewhat contracted. Physostigmin was instilled in both eyes and the eye which had been operated on was bandaged.

January 18, 8:30 a. m.: The bandage was removed. Anterior chamber reformed; cornea clear; pupil dilated ad maximum. T + 3, and vision equaled fingers at one foot. Hemorrhages were absorbed. A posterior sclerotomy was now made between the superior and external recti muscles under 10 per cent. cocain, and about 10 minims of a clear, straw-colored fluid escaped. The eye was very soft. Calcium chlorid in fifteen-grain doses was given every three hours, with a view of increasing the coagulability of the blood. In addition, nitroglycerin was given, 1/100 grain, four times a day.

January 19, 8:30 a. m.: T + 3. The eye was free from pain; the pupil fully dilated; the field good. The vision equaled fingers at one foot. Small hyphema present and the cornea was slightly hazy.

January 19, 2:30 p. m.: Hemorrhage disappeared. Dr. W. H. Wilder in consultation agreed with me in still holding out against an iridectomy. A combined paracentesis and posterior sclerotomy between the inferior and external recti muscles was made, under 10 per cent. cocain. Directly after the sclerotomy, bright red blood flowed into the lower part of the anterior chamber, appearing to come from the region of the iris root. The eye was very soft.

January 20, 8:30 a. m.: The bandage was removed. The cornea was clear; hemorrhage absorbed; pupil widely dilated; no pain.

January 21, 9 a. m.: The patient's condition was unchanged. The use of physostigmin, 3 grains to the ounce, was begun.

January 21, 2 p. m.: T + 3. The pupil was slightly contracted; now about 4 mm. in diameter; was still free from pain excepting after instillation of physostigmin. Blood pressure and coagulation time were taken by Dr. Adolph Gehrmann of the Columbus Laboratories, who found blood pressure 95 mm., pulse 80, coagulation time two to three minutes. The blood pressure was taken every day following for a week, and never went higher than 115 mm.

January 22, 8:30 a. m.: Condition was as noted yesterday afternoon.

January 23: The pupil about the same; tension slightly lower. Physostigmin was increased to 4 grains to the ounce.

January 24: Pupil was 4 mm., but there was a perceptible diminution in tension; cornea clearing.

January 25: The condition was unchanged.

January 26: The cornea was bright. The patient counted fingers at three feet; there was no change in tension from that last noted. Owing to the cloudiness of the vitreous, a view of the fundus could not be obtained.

January 29: The pupil was 2 mm.; tension + 1.

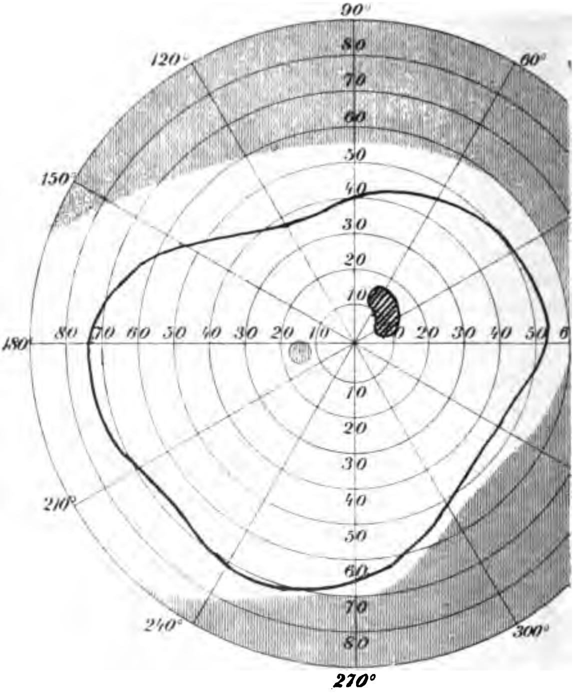


Fig. 1.—Field of vision for white (left eye), taken Jan. 30, 1908. Bright morning. Pupil 3 mm. in diameter. Disc, 1 cm.

January 30: The tension was normal; field was taken (Fig. 1) and the eye transilluminated for a possible tumor, but found negative. The patient counted fingers at one foot.

February 4: The eye was nearly white; the tension normal; vision, 6/60; the pupil 3 mm. in diameter. This condition remained up till February 11, 1908.

February 12, 8:30 a. m.: At about 4:30 p. m., February 11, there was some pain in the eye, which continued during the night, but the patient attributed it to the use of physostigmin. Examination now showed a new atack. The cornea was steamy; tension + 2; pupil about 4 mm. in diameter; the anterior chamber of same depth as its fellow. Iridectomy was now advised as a last resort, and the patient made aware of the possible danger. Operation was done under 10 per cent. cocain anesthesia, and a broad iridectomy performed with a Landolt keratome without a mishap.

February 13, 2 p. m.: The patient had been very comfortable; was free from pain. The bandage was removed; the cornea was clear; the anterior chamber reformed. Tension was slightly subnormal. The pupil was 4 mm.

February 16: Tension was normal; the eye quiet; physostigmin was reduced to 1 grain to the ounce.

February 17: The condition was the same. With the ophthalmoscope numerous large clots were seen floating in the vitreous, but nothing further could be distinguished; the patient was given syrup of hydriodic acid.

February 27: The condition was the same. The vitreous had cleared so that the location of the disc with vessels could be made out. The vision equaled 6/60.

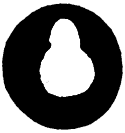


Fig. 2.—Anterior half of left eye as seen from behind (exact size).

March 1: I did not see the patient from February 27 until this day. Tension was normal, but there was a broken ring of pigment on the anterior capsule, and one corner of the nasal pillar of the coloboma adherent to capsule. Physostigmin was stopped.

March 11: There was no change in the eye until this day, when a dust-like exudate was found on Descemet's membrane. Tension was slightly above normal; cornea clear.

March 12: The patient had pain during the night. The cornea was steamy; tension + 2. The use of pilocarpin was begun.

March 13: Tension was normal. The cornea was clear; keratitis punctata well marked.

March 14: Up to this date the right eye had been absolutely free from any discomfort, but this day there was a fine ciliary injection, some lacrymation, and a feeling of discomfort. Tension was normal. On examination with a loupe, numerous fine dots were seen on Descemet's membrane, which stained deeply with fluorescin. Right eye vision equaled 6/5 minus. Accommodation was impaired. The disc was slightly hyperemic and the vitreous hazy. The left eye tension was normal. The vision equaled fingers at four feet. With these conditions, enucleation of the left eye was immediately advised and done under ether anesthesia.

March 14: Treatment was given, consisting of mercurial inunctions, pilocarpin sweats, sodium salicylate, dionin 5 per cent. in right eye.

March 28: Exudate was absorbing on Descemet's membrane; there were large masses of exudate in vitreous. The disc was slightly hyperemic. Tension was normal. Vision equaled 6/7. There was a large subconjunctival hemorrhage in stump of enucleated eye.

April 18, 1908: Right eye vision was 6/6 plus; a few very fine spots of exudate were still visible on Descemet's membrane, with the loupe. The vitreous opacities were smaller; eye accepted + 0.75 D sphere; vision equaled 6/6.

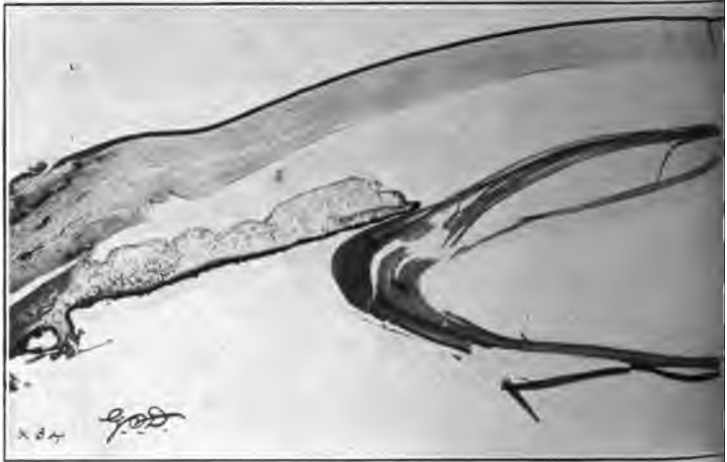


Fig. 3.—Section of left eye (photomicrograph, magnified 17 times). The upper limbus with the corneal incision and iridectomized iris lie to the right. One notes that: (1) The cornea is thinned; (2) the lens is thrown forward; (3) the chamber is shal-

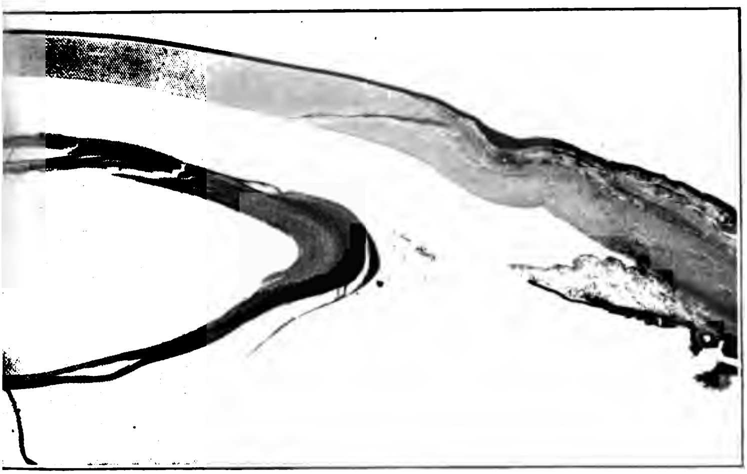
February 23, 1909: The external eye was normal; the vision equaled 6/5 —. Some vitreous opacities were still present and a few very fine dust-like opacities were still remaining on Descemet's membrane.

MICROSCOPIC REPORT

The eye was given to Dr. E. V. L. Brown for anatomic study, and he has submitted the following report:

"The specimen (Lab. No. 264) was fixed in formalin followed by Zenker's fluid, and was cut in an anterior and posterior half, the anterior half sectioned throughout the area of the coloboma and the posterior sectioned horizontally. Some 400 serial sections of each half were made and every fifth or tenth stained.

"Seen from behind, the anterior half presented the appearance sketched in Figure 2; the lens is clear, the pars plicata and corona ciliaris are *in situ*, the processes and plicæ apparently normal and regular in arrangement. An accurate sketch of the position and extent of the operative coloboma area was made, because, clinically, a perfect and entirely effective iridectomy had been performed. No portion of the root of the iris could be seen behind the opaque corneoscleral limbus, either before or after the eye was taken out; yet, as shown in the sketch, the gross specimen shows that a good 0.5 mm. of the root of the iris had not been removed by the operation.



low; (4) the sinus angles are open; (5) the trabeculum corneosclerale is infiltrated on each side; (6) the canals of Schlemm are open; (7) pigment epithelium is detached from the iris pupil zone and adherent to the lens capsule; (8) the iris stump above is 1 mm. long.

"*Microscopical Study.*—In a section through the center (section 205; see photomicrograph, Fig. 3) the curvature of the cornea and sclera are seen to be about the same. The cornea shows a tendency to collapse, and the center is slightly thinned to 0.5 mm. The epithelial layer is narrowed and elevated into empty vesicles in a number of sections; some of these (as section 110) have ruptured and left Bowman's membrane exposed. The outer cells are compressed and keratosed; the inner cell nuclei are thrown slightly forward or 'stilted'; many leucocytes are seen among the cells, esepcially in the upper portion of the cornea. Bowman's membrane is everywhere a trifle wider than normal and shows numerous delicate nerve canals as cross-striations. The body of the cornea is normal, although the very center takes a lighter eosin stain than usual. A narrow densely-nucleated scar, corresponding to the site of the corneal section for the iridectomy, lies at the upper limbus; it commences just above the end of Bowman's membrane and runs obliquely from without, inward and downward at an angle of 45 degrees from the end of Bowman's membrane to a point 2.3 mm. central to the upper end of Descemet's membrane. Here at its inner end the cut layers of the cornea gape slightly, so that the anterior chamber bays forward into the back part of the cornea. The defect has the form of a narrow wedge, and both lips are bordered by the curled-up ends of Descemet's membrane; the surface of the triangle is covered by a single layer of new endothelial cells directly continuous with those lining the anterior chamber. The anterior end of the scar grades off into a mass of proliferating endothelial and stroma cells just beneath the limbus epithelium. A closely knit matrix of new stroma cells has definitely healed together the anterior three-fourths of the wound. In more temporal sections (see section 185) the defect produced by the operation extends forward as a narrow sinus one-half the way to the surface of the cornea. But the lips of the wound anterior to the sinus are so firmly knit together that no 'filtration cicatrix' could have been present. In sections still more temporal (such as section 110) several well-filled vessels of small size are seen running through the scar and are surrounded by considerable numbers of round cells. In all sections through the scar the surface epithelium is normally reformed, but nowhere dips down into the wound tract; the final repair has been effected entirely by proliferation of the fixed corneal cells. The lower one-half of Descemet's endothelium is thinned and the cells in part disintegrated; many clumps of small round cells and pigment detritus are found on the posterior surface of the cornea; some sections (Cf. section 110) showing as many as six clumps in the lower angle of the anterior chamber.

"The conformation of the anterior chamber and the sinus angles deserve special description. The entire lens system and iris body have been displaced forward, narrowing the chamber as a whole; indeed, the anterior pole of the lens rounds forward through the pupil to such an extent that the space between the anterior capsule and the cornea is reduced to two-thirds of a millimeter. The plane of the iris below is correspondingly thrown forward, and makes an angle of 25 degrees with the transverse axis of the eye in comparison with a normal angle of about 15 degrees. Yet the sinus angles are patent in all sections both above and below, and the fluids of the chamber have free access to the surface of the trabeculum corneosclerale; the angle above is reduced to a narrow slit but is free from cells. The iris below measures 3.23 mm. from the pupillary margin to the circulus major, whereas the iris above has been in a large part cut away by the iridectomy; the iridectomy extends from section 49 temporal to section 213 nasal; the columns are nearly parallel and nowhere in contact with the wound. The stump lies entirely behind and peripheric to the corneal portion of the corneoscleral limbus, corroborating the statement made above that the iridectomy clinically was a very peripheric one; yet accurate measurement of the shortest portions of the stump shows that over 1 mm. of iris, measured from the circulus major to the cut end, is still present (section 143). As stated before, the angle does not show any acumulation of round cells or plastic material in any section; a heavy eosin stain does not bring out more fibrin or albuminous material than would be expected in aqueous reformed after evacuation. What fibrin is present is almost entirely in the lower half of the chamber and disposed along its walls.

"The sinus angle above is wider than that below; the cut end of the iris stump does not reach the equator of the lens anywhere and consequently the iris above has been displaced backward into a position that is even more nearly at right angles to the optic axis than in a normal eye. The iris below is thrown forward so as all but to touch the base of the cornea in many sections; indeed, some anterior root synechia to the back portion of the trabeculum corneosclerale has taken place in the sections which go through the angles just a little temporal to the iridectomy area; sections still more temporal are again perfectly free from any evidence of union between the periphery of the iris and the back part of the trabeculum corneosclerale. Both surfaces are perfectly smooth and sharply defined in all except a few peripheric sections (as, for instance, section 49) which show good-sized pigment masses.

"The anterior half of the iris, trabeculum corneosclerale and canals of Schlemm are infiltrated with round cells; those in the iris are almost exclusively of the plasma cell type and only moderate in amount; the posterior layers of the iris do not show any considerable amount of cell increase; the stroma pigment cells are considerably disturbed toward the iris root both above and below. In most sections the infiltration about the canals of Schlemm is so dense or the cells so changed that only round heavy-staining nuclei are seen and the type of cell can not be made out. Despite this, the lumina of the canals are free and lined by a well-preserved endothelium in most sections and filled with blood in many; the position of the canals can always be readily made out. Further evidence of plastic inflammation of the iris is furnished by deposits of pigment detritus, clumps and short strips of pigmented cells on the anterior surface of the lens capsule. Their exact location and the relations to the pupil border of the iris are of great importance in any attempt to establish an order of pathogenesis in the findings.

"In the sections which go through the temporal (sections 60-120) or nasal (sections 144-205) portion of the lens capsule the pigmented cells lie in about the center of the portion of the capsule shown and these (vertical) sections go through two areas, each about 5 mm. wide, in the horizontal plane. The central sections (120-144) show no deposits on the center of the part of the lens capsule concerned, but do show deposits more toward the equator of the lens; these central sections go through a pupil area of about two-thirds of a millimeter in diameter. These evidences of posterior synechia, therefore, extend through an area ·5 mm. in width and are absent through a zone of two-thirds of a millimeter corresponding to the size of a normal pupil. Furthermore, there was no actual posterior synechia present at the time the eye was removed, because no sections show adhesion betwen the iris and lens; the slight expanse of the iris below (only 3⅓ mm. overlap the lower equator of the lens for a millimeter in section 133) means that the pupil was well dilated at the last. One may conclude, therefore, that there was first formed a central posterior synechia in a zone corresponding to the position of the sphincter at a time when the pupil was small, and that this was later broken up by dilatation of the pupil and did not reform. These facts are in keeping with the clinical history.

"The ciliary body is markedly atrophic; the processes are all directed forward and much flattened down against the muscular portion of the body. The muscle has lost its triangular form and instead of a prominent angle on the inner anterior face, it presents a very flat curve extending forward from the region of the equator of the lens forward to the canal of Schlemm. No cell increase is found in any part of the ciliary body, and the stroma does not show any increase of connective tissue; a few of the processes are matted together at their bases; the apices are all free and there are no cells, fibrin or new tissue in the zonula ciliaris or anterior vitreous.

"The chorioidea is *in situ* and absolutely without any evidence of active or past inflammation, except where one of the sclerotomies was performed. Here the scleral wound is well healed externally, though gaping slightly internally; the cut ends of the chorioidea and retina are closely in apposition or slightly prolapsed in to the inner angle of the wound; the chorioidea shows round-cell infiltration for one-half to three-fourths of a millimeter only on each side of and in the wound and in a few sections only (section 289 and section 320); here the sclera is slightly ectatic. The chorioidea is not at all thickened by the process, the infiltration is never more than eight or ten cells in width from within outward, no epitheliod cells can be found, and the whole process apparently represents only normal wound healing after posterior sclerotomy. The retina in this area is atrophic and fused with the chorioidea into a dense atrophic scar permeated by broken-down pigment epithelium. Several *Drusen* bodies are found in the anterior part of one section, 380.

"Elsewhere the retina was detached in preparation and shows no evidence of inflammation aside from mantles of round cells about some vessels; it is markedly thinned. The vitreous is slightly shrunken in bulk, but otherwise quite normal aside from a considerable amount of fresh blood near the ora serrata below.

"One hundred and ninety serial sections through the portion of the optic nerve immediately behind the disc and including the entire course of the central vessels within the nerve show no evidence of inflammatory or atrophic changes in the nerve substance, and no changes in the blood vessels.

ANATOMIC SUMMARY

"We have therefore an active chronic iridocyclitis, healed operative corneal and scleral wounds, and an operative coloboma of the iris above. The eye shows effects of increased intraocular pressure in the displacement of the iris and lens system forward, in the flattening of the ciliary body and in the marked atrophy of the retina. No anatomic or histologic evidence of sympathetic inflammation is present. The presence of the chronic iridocyclitis throws doubt on the clinical diagnosis of primary glaucoma, and it is impossible to say from the anatomic findings that an anterior uveitis without clinical manifestations had not preceded and caused the glaucoma."

The conclusions deduced from the microscopic examination deserve careful consideration on account of their differences from the clinical findings. First, as to the non-existence of anatomic or histologic evidence of sympathetic inflammation: Although the changes in the iris, ciliary body and chorioid, as described by Fuchs, were absent, nevertheless the clinical symptoms were those of a sympathetic inflammation. Whether this was due to some agent entering the exciting eye as the result of the iridectomy, paracentesis or sclerotomy, or to the same thing which caused the glaucoma, whatever that may have been, I am unprepared to say. Until the pathologic anatomy of infiltrative-proliferative uveitis as described by Fuchs is found in all cases, and more firmly established as a pathognomonic finding, our diagnosis must be based on the clinical manifestations of the disease. The mere fact that the anatomic findings in this eye show a chronic iridocyclitis does not prove that it preceded the glaucoma and made it a secondary glaucoma, since the clinical precipitates on the posterior layer of the cornea and posterior synechia were not present until eight weeks after the onset of the primary glaucoma; but they did follow just exactly twenty-eight days after the iridectomy, and may very well have caused it. The patency of the sinus angles in all sections, and the lumina of the canals of Schlemm, free and lined by a well-preserved endothelium in nearly all sections, is striking and deserves careful consideration. That a gross obstruction of the filtration angle at any point in the filtration zone accounted for the increased intraocular pressure is not at all probable, and the fact that another attack followed iridectomy seems to be both pertinent and conclusive. It is more reasonable to assume, according to Priestley Smith, Bulson, Tenny and Troncoso, inasmuch as primary glaucoma in a certain group of cases is dependent on disease of the blood or blood vessels causing an excess of fluid in the vitreous chamber, the exit of which is prevented by the swollen ciliary processes, or on alteration in the constitution of the intraocular fluids with failure in drainage, that the acute glaucoma here was dependent primarily on some vascular disturbance. As a result of this vascular disturbance, possibly due to some form of toxemia, there is diminution in the circulation of the blood favoring the occurrence of hemorrhages which result rather from transudation than from ruptured vessel walls.

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NODULAR OPACITY OF THE CORNEA

WITH SPECIAL REFERENCE TO ITS ETIOLOGY*

JOHN GREEN, Jr., A.B., M.D.
Ophthalmic Surgeon to the Social Service Hospital.
ST. LOUIS

The ophthalmic literature of the last thirty years contains occasional references to a rare ocular disease characterized by the presence of fairly well-defined opacities situated in the central portion of the cornea. These opacities vary in size and contour and lie in the otherwise relatively clear tissue. The condition was not recognized as a distinct malady until Groenouw, in 1890,[1] and again in 1898,[2] made it the subject of clinical investigation and pathologic study. Since then a number of observers have dealt with the condition and a few have subjected the diseased tissue to histologic examination.

Groenouw states that nodular opacity of the cornea begins with slight inflammatory signs, which are often so insignificant as to escape the notice of the patient. It attacks both eyes and is distinguished by the appearance of numerous, small, roundish or irregular, gray discrete opacities in the otherwise relatively clear corneal tissue. The larger masses, which are situated principally in the superficial layers of the center of the cornea, attain a diameter of 0.25 mm. The overlying epithelium is frequently elevated, but not vesicular. Under high magnification the corneal surface between the masses is seen to be very faintly cloudy.

Our knowledge of the condition was considerably extended by Fuchs[3] on the basis of eight carefully observed cases. He found that the trouble showed a special predilection for young men; (11 males, 1 female, in the 12 cases in the literature up to 1902). It was characterized by: (1) considerable roughness of the corneal surface; (2) gray, roundish or irregular opacities situated in the pupillary area and projecting above the corneal surface; (in general the larger irregular masses were surrounded by smaller roundish ones, the latter exhibiting a circular arrangement); (3) a faint diffuse opacity of the rest of the cornea which, under high magnification, resolved itself into very tiny punctations. The configuration of the opacities slowly altered in the course of years.

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[1] Arch. f. Augenh., xxi, 281-289.

[2] Arch. f. Ophth. (von Graefe's), xlvi, 85.

[3] Arch. f. Ophth. (von Graefe's), 1902, liii, 423.

The extreme rarity of this disease is attested by the fact that Fuchs encountered the condition only eight times in a material comprising upwards of 250,000 eye cases. At present writing the literature contains only 22 authentic cases. (Groenouw 2, Fuchs 8, Holmes-Spicer 2, Chevallereau 1, Krukow 3, Gillius 2, Wehrli 2, Treacher Collins 1, and Paderstein 1.)

SYMPTOMATOLOGY

The disease is ushered in by moderate photophobia, epiphora, and occasionally a foreign body sensation. There is not true pain but only a sense of ocular discomfort. These signs are accompanied by diminution of visual acuity. Throughout the long course of the disease there is a conspicuous absence of active inflammatory appearances, although now and again a transient redness, accompanied by photophobia, will manifest itself. The course of the disease is essentially chronic.

In view of its progressive character and rebelliousness to all therapeutic methods, the disease is one that deserves the careful attention of ophthalmologists. The final visual outcome varies from tolerably good vision to finger-counting.

DIFFERENTIAL DIAGNOSIS

Nodular opacity of the cornea is to be distinguished from a number of more or less similar conditions. Fuchs calls attention to some distinguishing features between this disease and superficial punctate keratitis. In nodular opacity the spots are compact and homogeneous. There are no inflammatory appearances and the clinical aspect may remain unchanged for years. In superficial punctate keratitis, on the contrary, the spots are made up of an agglomeration of the very finest dots. Inflammatory appearances are invariably present and the condition undergoes rapid changes. Von Reuss,[4] under the name of "keratitis maculosa," describes a disease consisting of disc-like masses surrounded by a ring occupying for the most part the periphery of the cornea, but also at times the center. Nummular keratitis (Stellwag[5]) consists of very large masses of a saturated yellowish tint lying mostly in the periphery of the cornea and in the superficial layers. Both types, in contradiction to nodular keratitis, offer a good prognosis. Grill-like keratitis is a condition which, in respect to its course, its situation in the center of the cornea and the elevation of the epithelium over the lesions, presents many points of similarity. It is distinguished by the very definite mesh-work or interlacing arrangement of the linear opacities. Fuchs suggests that nodular opacity bears some resemblance to certain cases of old eczematous keratitis in which a thinning of the epithelium over one or more of the denser opacities produces a bluish-white prominence of the corneal surface. According to Morax[6] the disease may be confused with syphilitic nodular keratitis, in which the infiltrations, either solitary or in groups, are of a grayish-yellow color. They develop slowly, last for several months and finally disappear, leaving only insignificant deep or superficial opacities in the cornea. Under the title "Spotted Family Degeneration of the Cornea," Fehr[7] describes a condition observed by him in two sisters and a brother. Each cornea showed a milky opacity in which scattered dots of greater saturation were distinguishable. Under magnification the diffuse opacity resolved itself into tiny punctations and the more saturated masses were seen to be due to the crowding together of the individual punctations. This condition is distinguished from nodular opacity by the fact that the periphery of the cornea is involved and that the corneal surface is smooth and shiny.

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[4] Wien. klin. Wchnschr., 1889, No. 34.
[5] Wien. klin. Wchnschr., 1889, p. 613.
[6] Encycl. Franç. d'ophth., v. 949.
[7] Centralbl. f. prakt. Augenh., 1904, p. 1.

HISTOLOGY OF THE AFFECTION

Fragments of the diseased tissue have been studied histologically by Groenouw,[2] Fuchs,[3] Chevallereau,[8] and Wehrli.[9] The only report based on the examination of the entire globe is the recent one of Paderstein.[10] Groenouw's specimens showed a foreign substance lying partly isolated and partly in large masses in the substantia propria of the cornea. Just beneath the epithelium the material appeared finely granular and exhibited a certain parallel or interlacing arrangement. It had the appearance of a foreign substance interposed between the corneal fibers. Adjacent to the deposits the corneal corpuscles were deformed and greatly increased in numbers. There was no change in the epithelium or Bowman's membrane. From the chemical reactions of the amorphous substance Groenouw was led to believe that it was of a hyaline nature. The presence of this substance he explains on two grounds: either it was a transformation product of the corneal substance or a foreign mass deposited in it. He admits the possibility of a combination of the two processes.

In Case 6 of Fuchs' series, a fragment of the corneal tissue from the pupillary area, 4 mm. in diameter and 0.25 mm. in thickness, was excised with a corneal trephine. The pathologic appearances, which were confined to the superficial layers of the cornea, were as follows: The epithelium covering the nodules was greatly thinned and the cells flattened. The more superficial corneal lamellæ were swollen and, at sites corresponding to the nodules, were separated. Between the superficial and deeper layers appeared a deposit of amorphous substance, which was not present beneath the nodules. The substance contained isolated corneal fibers and wandering cells. Bowman's membrane could not be seen. When stained with thionin those layers beneath the amorphous deposit took a violet color, instead of the usual blue-green of normal corneal tissue. There appeared then four alterations from the normal: (1) swollen appearance of the upper corneal layers throughout; (2) fibrillation of these layers in limited areas; (3) a deposit of amorphous substance; (4) a change of the underlying lamellæ. Fuchs assumed a metabolic change in the superficial layers leading to the formation of a pathologic fluid. This fluid brought about a swelling of the superficial layers, and, accumulating between the lamellæ, separated and finally destroyed them. He was unable to reconcile the anatomic findings in his case with those of Groenouw and others and was forced to adopt the hypothesis that etiologically and anatomically different processes gave rise to clinically indistinguishable corneal conditions. He concludes that the disease is due to a general, as yet unknown, nutritional disturbance of which the sole expression so far observed is the eye disease.

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[8] France méd., May 2, 1891.
[9] Ztschr. f. Augenh., 1905, xiv, 322.
[10] Klin. Monatsbl. f. Augenh., February, 1909, p. 156.

Chevallereau[8] found crystals of sodium urate lying between the superficial corneal layers.

Paderstein[10] reports the only histologic examination of the entire globe in a case of nodular opacity of the cornea. He found thinning of the epithelium and altered relations of the round and cylindrical cells. The space of the missing cells was filled with a homogeneous hyaline substance, which also appeared on or slightly separated from Bowman's membrane. In places the nodules appeared to be made up of layers of hyaline "like the flakes of an onion." Elsewhere the hyaline assumed a fibrous appearance. Bowman's membrane varied in thickness and was pressed together by the overlying nodules. In places a sharp differentiation between the hyaline and Bowman's membrane was not possible. The hyaline lay also on the basal epithelial cells. These, and to some extent other epithelial cells, had separated from neighboring cells. Their nuclei were partly normal, partly degenerated, and vacuolated. There were no pathologic changes in the corneal parenchyma. Paderstein believes that the basal epithelial cell layer was the primary seat of the affection, and outlines the series of pathologic events as follows: A degeneration of the epithelial cells resulted in the formation of hyaline. The nodules and projections were formed by agglomeration of degenerated cells. Between the cell groups, which had passed through a complete transformation, were scattered some in which the degenerative process was not completed. Thus are to be explained the cells and cell remains in the spaces between the layers of the deposit.

From the above descriptions it can readily be seen that the histologic findings, though presenting some points of similarity, are widely at variance in others. Entirely unrelated to any appearances described by other observers are the findings of Wehrli.[9] This author encountered the disease in two brothers, one of whom had passed through an attack of tuberculous peritonitis. He excised some of the nodules and introduced them into the anterior chamber of a rabbit. There was a sharp reaction with the appearance of a transient yellowish exudate around the fragments, but nine months later the rabbit's eyes were apparently normal. The histologic findings included enlargement of a portion of the fixed corneal cells and increase and regressive processes in a number of the same. The large cells were arranged in groups and bore a striking resemblance to the histologic disposal of the cell nests of epitheloid nodules of tuberculous or syphilitic granulomata. Furthermore, there were found large cells with several nuclei, similar to giant cells. The connective tissue fibers had a sort of cicatricial character. Those next to the so-called tubercles were granular and homogeneous. An amorphous substanc deposited in the superficial layers was regarded by Wehrli as necrotic débris from the destruction of tuberculous nodules. By the Ziehl-Nielson method Wehrli found, after long search, two apparently segmented, slightly curved bacilli, coloring faintly red, which he believed to be tubercle bacilli.

In view of the extraordinary rarity of the affection, and also of Wehrli's dictum that every observed case deserves recording, I venture to present the following typical example of nodular opacity of the cornea:

REPORT OF CASE

General History.—R. V. O., aged 27, male, married, came under observation Oct. 30, 1908. His mother died of tuberculosis of the lungs at the age of 40, the patient being 10 years old at the time. A maternal aunt also died of the same disease. As a young boy the patient was perfectly healthy. At the age of 9, he fell out of a swing and landed on his back. The following day a "ridge" appeared around the waist, not accompanied by pain. This remained and two or three weeks later a swelling appeared on the left hip, which was lanced and discharged pus. Other swellings of a similar nature appeared to the right of the sacrum near the coccyx and over the left gluteal muscles. All these swellings were lanced and discharged pus for several years, finally healing when the patient was about 17 years old, leaving large deep scars. For a time the left knee was flexed and the patient had to use a crutch. There was no pain in the joints at any time. At the age of 16 the patient passed several kidney stones, preceded by severe pains in the lumbar region. Six years before he came under my observation he had a severe attack of measles and gonorrhea three years later—mild attack, no complications. He positively denied syphilis. For the past ten years he had been exceedingly healthy, though troubled with a winter cough. His average weight was 135, maximum 150 (present weight.)

General Examination.—I am indebted to Dr. L. M. Warfield for the general physical examination. This showed a puckered scar on the left groin three inches in length, one on right groin, one large deep scar opposite the left trochanter, one on each side of the upper part of the sacrum, small one to the right of the coccyx, one puckered scar below the edge of the gluteus maximus. At the lowest lumbar spine the bones were thickened; there was no pain, no limitation of motion, slight limitation of adduction. The lungs and heart were normal. The scars were typical tuberculous cutaneous scars. There was no evidence of syphilis.

Ocular History.—About three years before the time of examination the patient noticed a sandy or gritty sensation accompanied by slight epiphora and photophobia and found that his sight was growing dim. There was no noticeable redness and at no time any true inflammatory signs. A year later he consulted on oculist who used various local medicaments and gave mercury by the mouth. Vision grew slowly worse, though there were transient periods of slight improvement.

Ocular Examination.—There was no conjunctival or ciliary congestion in either eye. R. V. 18/150. L. V. 18/96. Examination with the binocular loupe disclosed five or six, very irregular, rather ill-defined grayish masses occupying the center of the right cornea. These masses were, in general, grouped in the form of an ellipse, the center of which was occupied by a nebulous area. The left cornea presented much the same appearance, the opacities being of irregular shape, rather more sharply defined than in the right eye and arranged in the form of a flattened ellipse. The center of the ellipse was rather clearer than in the right eye. With the aid of the Zeiss corneal microscope a very beautiful picture presented itself. In the right eye (Fig. 1), the irregular masses appeared fluffy, like cumulus clouds, with ill-defined margins, shading into relatively clear cornea. The masses could not be resolved into separate dots, but appeared quite homogeneous. Where two separate masses lay near together there was a decided tendency for them to merge or coalesce. The tissue occupying the center of the ellipse was distinctly nebulous. Here and there, the epithelium over the nodules appeared elevated, so that it formed a tiny facet and reflected a laterally projected pencil of light. Outside the involved central area the cornea was very faintly nebulous and this cloudiness extended right up to the corneal margin. The lesions were situated in the superficial layers, but here and there appeared to dip well back into the corneal stroma. The iris showed no alteration. In the left eye (Fig. 2) the masses were somewhat better defined than in the right, i.e., the line of demarcation between the individual nodules and the contiguous tissue was better defined. The nodules themselves were not as fluffy as in the right eye but appeared homogeneous. The space between the masses was rather less nebulous than the corresponding space in the right eye. The epithelium over the nodules was elevated and irregular. The periphery of the cornea contained an extremely delicate gray opacity which resolved itself into the finest of dots. The fundus of each eye was normal. Tension normal; pupils equal; pupillary reaction normal both to light and convergence. The general appearance of the cornea is well illustrated by the accompanying pictures. These were drawn, however, some three months after treatment was begun and do not show the distinct internodular opacity in the left eye visible at the first examination. The history of the case, together with the quite typical appearance, left no doubt as to the diagnosis of nodular opacity of the cornea.

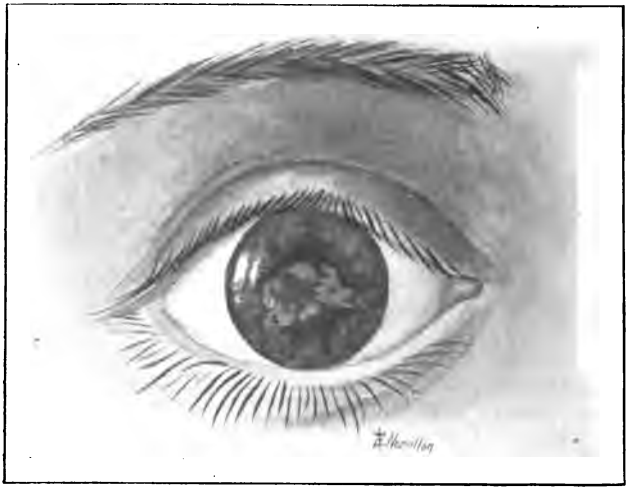
Fig. 1—Nodular opacity of the cornea, right eye.

Treatment and Course of Disease.—Bearing in mind the clear tuberculous family history, and the unquestioned tuberculous nature of the old cutaneous ulcerations, I was impressed with the possibility that the eye lesion might be of the same nature. It was, therefore, decided to try the effect of a diagnostic injection of tuberculin.

October 31: At 3 p. m., 3 mg. of old tuberculin were injected into the upper left arm. The following day, November 1, 11 a. m., temperature was 98.4 F. There was very slight redness and soreness at the point of inoculation, with some palpable thickening. At 8 p. m., temperature 99 F.

November 2: Arm quite sore; 3 mg. old tuberculin were injected in the right arm.

November 3: Both conjunctivæ were injected and there was some ciliary congestion. Patient complained of much discomfort, increased photophobia and stickiness of the lids. On examination with the corneal microscope the nodules in the right eye especially were denser and more fluffy than at the first examination. R. E. V. 18/192. L. E. V. 18/96.

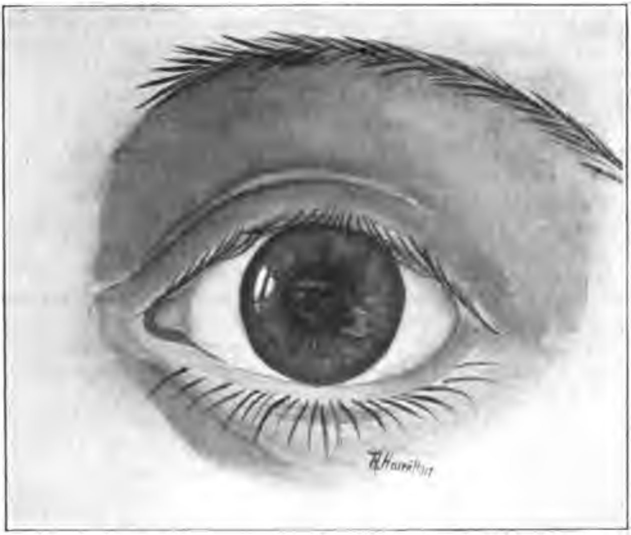

Fig. 2.—Nodular opacity of the cornea, left eye.

November 5: Both arms were found to be indurated at the site of the injections. Three mg. old tuberculin were given.

November 6: Patient stated that he had had a bad night; severe headache; now felt quite sick. At 10 a. m., temperature 99.4 F. Large induration at point of inoculation. At 4 p. m., temperature 100.6 F. R. V. 18/75. L. V. 18/38. The corneal microscope now showed a distinct thinning of the cloudy nodules in the right cornea and a diminution in the internodular opacity. The left eye showed a very distinct diminution in the internodular opacity but no noticeable change in the nodules themselves. The patient expressed his surprise at the sudden improvement in vision.

Diagnostic tuberculin injections were suspended and the patient was not seen again until November 12, on which date therapeutic doses were instituted, beginning with 0.01 mg. of old tuberculin. At intervals of three or four days old tuberculin was injected, increasing 0.01 mg. at each dose.

December 3: The dosage was 0.05 mg. and, for the first time, evoked some soreness at the point of injection.

The dosage was reduced to 0.02 and gradually increased at intervals of a week to 0.03, 0.04 and finally 0.055 mg. when treatment was suspended. The therapeutic injections between Nov. 12, 1908, and Jan. 13, 1909, numbered fifteen. At each visit the corneæ were very carefully examined with the corneal microscope and it was possible to note a gradual diminution in the internodular opacity, especially of the left eye.

On March 1, 1909, the masses were decidedly less dense and in the right eye were more sharply delimited than at first. This change in the appearance was accompanied by gradual improvement in vision so that at this date R. V. 18/60. L. V. 18/30. The patient was much gratified with the improvement in vision as he had about made up his mind that he was going blind. Local treatment has been confined to bland collyria and (for a week only) dionin 3 per cent solution. This was suspended, as it provoked a good deal of discomfort. I was anxious to secure a fragment of the cornea for histologic examination but the patient declined any operative interference whatsoever.

I have later learned that the patient's sister, residing in Kentucky, has had defective vision for a number of years. To a letter requesting particulars of the malady she replied that she had been troubled with her eyes for five years. There had never been any inflammation, the main diffculty being defective vision. She had tried several pairs of glasses but found none that helped. "Over the sight of the eyes are several white specks about the size of a pin-head."

We have, then, in a patient with a very clear family history of tuberculosis, who himself had passed through a severe cutaneous tuberculous affection, a typical nodular opacity of both corneæ. The absence of any etiologic factor aside from tuberculosis which might conceivably have given rise to such a process, the general and local reaction to the diagnostic tuberculin test, the lessening of the density of the nodules, the clearing of the internodular opacities and notable improvement in vision under tuberculin therapy, lead me to the belief that this case is an attenuated tuberculosis of the cornea.

Writers subsequent to Wehrli have been disposed to ignore his findings and conclusions, seeming tacitly to assume that because they did not coincide in any particular with those of other observers they must be faulty. In the light of my own case such a point of view seems to me not justified.

In the present state of our knowledge with regard to this peculiar affection, we must adopt the view that cases of nodular opacity of the cornea, clinically indistinguishable, may differ with respect to their etiology and histology. It seems proper to regard the cases of Groenouw and Fuchs as instances of a general nutritional disturbance of which the sole expression is the corneal lesion. On the other hand, the histologic findings of Wehrli and the diagnostic and therapeutic efficiency of tuberculin in the case here reported seem to indicate that some of these cases may be, as Wehrli has pointed out, examples of attenuated tuberculosis of the cornea.

626 Metropolitan Building.

MEMORANDA

PRACTICAL IMPORTANCE OF HYPERPHORIA

IN PRESCRIBING LENSES FOR USE AT PARTICULAR DISTANCES AND IN DIFFERENT DIRECTIONS *

M. D. STEVENSON, M.D.
AKRON, OHIO

Hyperphoria when the head is in the primary position and the test object on the line of intersection of the extended median and horizontal fixed planes of the head (Savage), has received considerable attention by many able investigators. This paper will deal more particularly with hyperphoria at the reading distance and at the reading angle. The prescribing of prisms to correct hyperphoria is of great importance. The correction of 1 degree of hyperphoria, constant at a series of examinations, will give in the average case better results than the prism correction of a very much greater degree of exophoria or esophoria. Normal eye muscles have difficulty in overcoming a 3.5 degree prism base up or down, while adducting power of 50 degrees is often found. Normal eyes have vertical equilibrium. If a series of examinations shows 0.5 degree of hyperphoria, decentering of the lens or the introduction of a prism is usually advisable.

The degree of hyperphoria is often extremely variable at different examinations and even at the same examination. Sufficient time should be given for the patient's eyes to be disassociated, as those who have a strong fusion faculty may at first attempt to combine the two images, even if they are unlike, as the light and streak with the Maddox rod. In some cases the eyes are very loth to disassociate themselves and to rotate independently. A first hurried examination

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may show no heterophoria but later examinations an increasing amount. The screen, parallax and other tests are sometimes necessary. Cycloplegics by their effect on accomodation, and I think, by direct action on the external muscles, sometimes seem to aid in unmasking latent heterophorias.

After determining the proper lens for each eye separately and after repeated examinations of the hyperphoria have shown a nearly constant amount, the prism is tested in the properly placed and centered trial frame while the patient looks at the distant test chart. Patients will frequently at first reject a much-needed prism placed for only a short time in the trial frame. It is necessary to take sufficient time, that the eyes may adjust themselves to the new conditions. When needed the patient will prefer to retain the prism, as its removal necessitates quite an effort for adjustment. It is often helpful to learn in which part of the test, putting on or taking off the prism, the patient experiences greater difficulty or requires more time to make the adjustment of his eyes. If it takes longer to adjust the eyes on removing the prism it is very likely that a prism is required.

If the hyperphoria found is less than 2 degrees, I usually prescribe the equivalent in degrees by decentering or prisms. When more than 2 degrees is present, I depend on comfort tests of prisms in the trial frames and do not, like many, always undercorrect. However, a 2.5 degree prism is usually selected for 3 degrees of hyperphoria and a 3 degree prism for 4 degrees of hyperphoria. There can be no fixed rule; each patient must be carefully tested. Hyperphoria of over 6 or 7 degrees, which is usually non-comitant or changeable (Fuchs), can seldom be comfortably corrected by prisms. Low degrees often occasion more discomfort and fatigue than high degrees because no attempt is made to overcome the latter. My operative experience has not been sufficient to enable me to determine definite policies in these cases. Prism exercises are sometimes helpful.

Many observers, recognizing the close relationship between accommodation and convergence, have studied exophoria and esophoria not only at the distance but in accommodation. It has been found that the excess or insufficiency of convergence (so-called), especially that found when fusion is entirely prevented, accommodation alone being active, varies greatly from the degree of esophoria or exophoria for the distance. A separate test is advisable before prescribing lenses for the reading or any special distance. Likewise hyperphoria for the near may vary from that at the distance as illustrated in a case by Theobald. Clinically it is more difficult to overcome a certain degree of hyperphoria for near than the same amount for distance. Not only is hyperphoria sometimes found to be variable at different distances, but the degree may also vary if the head be tilted up or down so that the eyes in looking at a distant fixed light are compelled to rotate in a different plane. Many of these cases are partial paralyses of the vertically acting muscles. Fuchs believes that many cases of hyperphoria are the result of paralysis. He classifies heterophoria as non-comitant, changeable, and comitant, also as periodic or continuous for near and far; Maddox classifies it as paretic and simple or con-comitant. In a considerable number of cases the hyperphoria varies, sometimes several degrees, depending on whether the eyes are in the primary plane or looking upward or downward. If hyperphoria at the distance sometimes varies in different planes, it would seem reasonable to suppose that a variation could also occur at the reading distance.

I have had an instrument made by the DeZeng Standard Co. of Philadelphia, to test the hyperphoria for near at various angles indicated on the instrument. The area of light through ground glass is controlled by a rotating disc with holes of various sizes. The light can be moved to any desired distance from the eyes. The Stevens phorometer in connection with the Maddox rod is employed so that the amount of heterophoria is easily indicated. If an amount in excess of that registered on the instrument is present, a clip is provided in which an extra prism from the trial case may be placed. The instrument may be used for distant muscle testing by using a distant light, it being unnecessary to remove the small lamp which, however, can be easily done. The convergence near point for a light may also be easily tested by this instrument on which the meter angles of convergence are also indicated. The lamp may be removed from the shaft of the instrument and used to examine the field of projection when the media are not clear, e. g., in cataract cases.

At the distance the primary horizontal plane is the most important. The best plane for near testing corresponds with that in which the patient usually directs his eyes in near work. This varies, generally in making an angle of about 30 degrees with the horizontal plane. In quite a number of cases the hyperphoria at this angle, more or less constant in several examinations, varies several degrees from that found in the horizontal plane in near or distant tests. As would be expected in paresis, the hyperphoria sometimes changes if the instrument is moved from side to side. Some patients with orthophoria at the distance show two, three or four degrees of hyperphoria at the reading angle. Right hyperphoria at one angle sometimes becomes left hyperphoria at another. Patients will often suffer discomfort if they do not have prisms of different strength in their lenses for distance and for reading. Many such can not wear bifocals (unless perfection with appropriate prisms) because the hyperphoria is not the same for the distance as for near. Some with their hyperphoria properly corrected in separate lenses for near and far are greatly benefited.

It is also important to consider the prismatic effect of lenses when the eyes look through them in different directions. Duane states that he has found hyperphoria present in 15 per cent. of low cases of anisometropia, and in 32 per cent. of high cases. Bruner has found hyperphoria in 30 per cent. of cases of anisometropia. Imagine a patient with anisometropia with a nearly plane lens before one eye and a strong sphere or a strong cylinder at axis 180 before the other. In such a case without hyperphoria in the primary plane, if the patient moves his eyes behind the lenses from the primary to the reading plane or *vice versa*, he has to overcome in all positions but one, a large degree of what may be termed artificial lens hyperphoria, which is often very distressing and one of the common reasons why it is difficult to give these patients relief. Separate distant and reading lenses or half grab-backs or grab-fronts with correct centering for a particular plane should more often be prescribed in anisometropia and antimetropia. If the lenses are strong great care should be used to see that they are properly centered. It is often easy to bend the frames up on one side and down on the other to center the lenses better for distant objects. In cases of anisometropia with proper lenses and frames before the eyes, I teach the patient by having him tilt his head up and down, that there is only one easy position in which to hold his head in order best to see distant objects. He is directed to hold his head so that it will be easiest for his eye muscles. The person receiving reading lenses of different strength is taught that there is not only a proper distance for his close work but also that it should be held at a particular angle. An anisometropic patient whose lenses are quite different must learn how to hold the head and the best position for the object looked at if he is to derive the greatest benefit.

Many who use bifocals, since they are often compelled to look through their lenses at a strong angle, are made uncomfortable, not only by the changed astigmatism, but also because the hyperphoria, artificial or true, at that angle is not considered. I now test all patients at primary and reading angles without lenses and with them, and as a result sometimes prescribe two pairs or perfection bifocals containing different prisms where I used to prescribe unsatisfactory cement bifocals. Since hyperphoria is changeable, its degree should be retested at intervals, especially if there is any discomfort in using the lenses.

Before this Section, it is unnecessary to be specific in outlining a policy in these varied and changing conditions. Each case must be a law unto itself, but this changing true or artificial hyperphoria with or without lenses deserves more attention.

Everett Building.

MEMORANDA

A CLINICAL INVESTIGATION ON THE RELATIONSHIP OF TUBERCULOSIS TO CERTAIN DISEASES OF THE EYE *

GEORGE S. DERBY, M.D.
BOSTON

AND

THOMAS H. AYER, M.D.
WESTBORO, MASS.

We wish to present for consideration a summary of the examination of ninety-two cases in which the ocular disease suggested a tuberculous origin. In selecting these patients for investigation our suspicion of tuberculosis rested on the clinical type of the disease process in the eye. The study was carried out during the five months (October, 1908, to February, 1909) in the outpatient clinics of the Carney Hospital and the Massachusetts Charitable Eye and Ear Infirmary, and we are much indebted to the staff of the latter institution for allowing us to make use of their material.

The plan of work was as follows: Having selected our patient, a careful record of the family history, personal history and present illness was made. The cutaneous test was then performed on the upper arm, for which at first we used a 25 per cent. solution of old tuberculin supplied by H. M. Alexander & Co. More recently we have discarded this for the full strength tuberculin as recommended by von Pirquet in his last publications. In all patients a control vaccination was made. The patient was seen after forty-eight hours and, in case of a negative result, watched until the possibility of a reaction was over.

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Following the vaccination, our patients were referred to the medical clinic of the Carney or to that of the Massachusetts General Hospital with the request in each instance that special attention should be paid to the question of an active or inactive tuberculosis. We wish to express our obligations to the members of the staffs of those two institutions who aided us in this connection.

In a few instances in which a definite diagnosis was of great importance and no confirmatory signs elsewhere could be obtained, the patient was taken into the hospital and the subcutaneous test applied for the purpose of producing, if possible, a local reaction.

In tabulating our results we have recorded our medical examinations as positive whenever the consultant reported the case as suspicious. As a matter of fact, there are but few cases in our series in which an absolutely definite diagnosis of tuberculosis elsewhere could be made, and those are, first, the ones in which there was a positive local reaction to the subcutaneous injection of tuberculin; and, second, a few cases of tuberculous adenitis or tuberculosis of the larynx and one of lupus of the nose. No bacilli were found in the sputum of any patient, which only serves to emphasize the early stage of the pulmonary process. In these patients the family history was recorded as positive if there was reasonable evidence of a tuberculosis affecting a member of the patient's immediate family.

Our cases may be roughly divided into two groups, in one of which the ocular inflammation was superficial, and one in which the deeper structures of the eye were involved.

GROUP I

The first group is made up of the so-called phlyctenular cases. We have fourteen cases of phlyctenular conjunctivitis with an average age of 14½ years.

In 3 of these (21 per cent.) the family history was positive. Only 1 patient (0.7 per cent.) showed an elevation of temperature. Nine (64 per cent.) gave positive physical signs. In 7 these signs were in the lungs, while 1 had a tuberculous adenitis and 1 a lupus of the nose. The cutaneous reaction was positive in 11 (78.5 per cent.).

The second division of the first group contains the so-called cases of phlyctenular keratitis (keratitis eczematosa, scrofulous keratitis), and we regret to classify them under so unsatisfactory a term; for in but few of them were actual phlyctenules observed. They are rather the cases of recurrent superficial inflammation of the cornea, with or without vascularization, occurring usually in children and young adults of unhealthy appearance. We have 52 such cases, with an average age of 9½ years; the youngest 1 year; the oldest 27. In this group the family history was positive in 13 (25 per cent.). The temperature was elevated in 25 (48 per cent.). There was positive physical fiindings in 23 (44 per cent.). In 17 of these the lungs were suspicious. Five had a tuberculous adenitis, 1 a tuberculous empyema, 1 a tuberculous larynx, 1 a tuberculous spine. In 48 cases (92.3 per cent.) the cutaneous test was positive.

It may be noted here in this connection that in a series of 43 phlyctenular cases published last year by one of us[1] 88 per cent. of patients reacted to tuberculin, and in the same paper attention was called to 23 similar cases tested subcutaneously by Stock with an exactly similar percentage of positive reactions. Fifty-nine of the 66 cases of this present series gave a positive cutaneous reaction (89 per cent.), a remarkably close approach to the former figures.

GROUP II

Leaving now for the moment the group of superficial inflammations, we have 26 cases in which the deeper ocular structures were involved. These are made up of 6 cases of scleritis, 6 of interstitial keratitis, 4 of uveitis, 7 of sclerokeratitis and 3 of keratoiritis. As a matter of fact all these cases may be placed together, for although they have been classified anatomically according to the location of the chief process, yet usually during the course of the disease neighboring tissues became involved.

Of our 26 patients the youngest was 6 years; the oldest 49; the average age was 26⅓ years. The family history was positive in 5 (19.2 per cent.). The temperture was elevated in 12 (46 per cent.). The physical signs were positive in 18 (69 per cent.). In 15 of these the lungs were reported as "suspicious," while in 3 there was a tuberculous adenitis. The cutaneous reaction was obtained in 24 (92.3 per cent.). It may be of interest to combine the figures of the first group and compare them with those of the second.

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[1] Derby, G. S.: The Increasing Importance of Tuberculosis as a Cause of Ocular Disease. The Newer Methods of Diagnosis and the Treatment, Arch. Ophth., 1908, xxxvii, No. 5.

GROUP I (SUPERFICIAL OCULAR INFLAMMATIONS) AND GROUP II (INFLAMMATIONS INVOLVING DEEPER STRUCTURES OF THE EYE)

FAMILY HISTORY

Group I: 66 cases. Positive in 16, or 24.2%.
Group II: 26 cases. Positive in 5, or 19.2%.
Whole Series: 92 cases. Positive in 21, or 22.8%.

TEMPERATURE

Group I: 66 cases. Elevated in 26, or 39.3%.
Group II: 26 cases. Elevated in 12, or 46.1%.
Whole Series: 92 cases. Elevated in 38, or 41.3%.

SUSPICIOUS SIGNS OF TUBERCULOSIS

(Lungs, glands, local reaction to subcutaneous test, etc.)
Group I: 66 cases. Positive signs in 32, or 48.5%.
Group II: 26 cases. Positive signs in 18, or 69 %.
Whole series: 92 cases. Positive signs in 50, or 54.3%.

REACTION TO TUBERCULIN

Gorup 1: 66 cases. Positive in 59, or 89.3%.
Group II: 26 cases. Positive in 24, or 92.3%.
Whole series: 92 cases. Positive in 85, or 90.2%.

In fifty cases out of a possible ninety-two there were signs pointing to a tuberculosis outside the eye, and as pertinent here we quote from the paper previously referred to:

> Since at autopsy tubercular lesions, healed or active, are found in from 40 per cent. to 90 per cent. of subjects, it follows that only in a small percentage of cases, most of them of a severe type, is the diagnosis made during life. There are then many mild cases, some of which are not seen by the medical man, and others in which the signs and symptoms are not clear enough to render the diagnosis possible. Most of these patients get well, and only in the event of autopsy is the disease manifest. In a large percentage of cases where it has been possible to make a definite diagnosis of tuberculosis, the disease has been in an advanced stage and cure has been uncertain. This is especially true of pulmonary tuberculosis, in which the finding of bacilli in the sputum clinches the diagnosis, but means advanced disease. I believe the same conditions exist in regard to ocular tuberculosis. Formerly only the hopeless cases, leading to the destruction of the eye, were recognized, while now we are beginning to recognize the milder types which are curable.
>
> If tubercular disease of the eye is as prevalent as my figures tend to show, it will lie in the power of the ophthalmologist to

take a more active part in the fight against tuberculosis than he can at present. We see hundreds of these cases in our clinics each year. My small series shows that many have signs pointing to tuberculosis in their lungs or elsewhere. These are the incipient cases that the tuberculosis expert wishes to reach. Even if the patient shows no general signs of the disease himself, he may be the means of directing attention to the whereabouts of an active focus of tuberculosis in the community.

Our present study tends to confirm these views. Furthermore, if we should search in these cases for signs of the so-called scrofulous diathesis we should find them, as many investigators of phlyctenular disease have shown, in a much larger number of patients, 85 to 90 per cent. As time goes on the identity of scrofula with tuberculosis is becoming more widely accepted. As long ago as 1904 Behring stated:

> Candidates for tuberculosis owe their much-discussed predisposition to an infantile infection with tuberculous virus. The practicing physician will before long become convinced that human pulmonary tuberculosis is no more nor less than the final stage of a tuberculous infection dating from infancy, which infection in childhood manifested itself in the signs of a relatively latent scrofulosis.

We believe that for the good of the community, if not for the individuals themselves, these eye patients should be given a thorough physical examination as a routine procedure. We think it likely that the deep and severe tuberculous infection of the eye are often the final stage of a tuberculous infection acquired in childhood and manifested at that time in the form of a phlyctenular process.

It is important that the significance of these forms of ocular disease should be brought to the attention of the opthalmologist and to the medical profession in general.

TUBERCULIN FOR DIAGNOSIS

In our work the use of the subcutaneous test for diagnosis has been found impracticable except in a small portion of the cases. It is impossible in large clinics to obtain the requisite number of hospital beds and it is very difficult to induce the patient to give up a sufficient amount of time for the test. Then, too, the moral effect of a positive reaction with marked general disturbance is often disastrous and has in our experience caused the patient to refuse subsequent tuberculin treatment. Furthermore, the anticipated local reaction may not be obtained. We believe in confining the subcutaneous test to those cases in which the diagnosis can be made in no other way and in which a diagnosis is essential is an intelligent course of treatment is to be prescribed. We have discarded the conjunctival test as being occasionally productive of disastrous results and as being but little more convenient than the cutaneous. We do not believe that there is as yet sufficient evidence of the superiority of the former test as indicating an active tuberculous focus. There can be no doubt that a negative cutaneous test is of far greater importance than a positive one. A positive reaction can add but little to the diagnosis of the ocular disease while as a result of two negative cutaneous tests tuberculosis may, with reasonable probability, be excluded. We have performed the latter test on nearly 200 patients and have never seen harm result from it.

TREATMENT

Although not included in the title of this paper, it may not be amiss to take up the question of treatment.

We reiterate the belief expressed by one of us previously[1] that these patients as a group are best handled by means of the tuberculosis class. In our work we have seriously felt the need of such organizations, and up to within a few months have had to refer our patients for treatment to the classes of other institutions. Three months ago we assisted in establishing such a class at the Carney Hospital for the treatment of all varieties of tuberculosis and in January a class for ocular tuberculosis alone (of which one of us is in charge) was financed by certain generous private individuals and was started at the Massachusetts Charitable Eye and Ear Infirmary. This we believe to be the first of its kind.

Our machinery there consists of an ophthalmologist, an internist, a social worker and a nurse. Patients are referred from the clinics to the class for the investigation necessary to diagnose, and when this is positive and the patient's case is a suitable one for class treatment he is registered as a member. We have a weekly session, but certain seriously affected patients are seen more often and the patients with milder cases may be instructed to report less frequently. Each patient is given a book in which a minute record of his daily life may be recorded. He is weighed, his temperature taken, and, if indicated, a tuberculin injection is given. This is but a part of the treatment, however, for it is most important to reach these patients in their homes and to exercise a strict control over their manner of living. Our treatment differs but little from that of the general tuberculosis class, except that our cases are usually a milder type of disease than the average one seen in the general class. We insist on rest during an active inflammation, and especially if the temperature is elevated during some period of the day, even if the rise be slight. The patients are taught cleanliness and how to get the maximum amount of fresh air. They have the usual antituberculosis diet and forced feeding. They are visited in their homes by our social worker as often as is necessary, and in this way we keep in close touch with them and do not have to depend on their own statements.

The severity and duration of the treatment needed varies markedly with the individual case. Most of the patients in phlyctenular cases soon recover and remain well if they contiue to live in good hygienic surroundings. We have them report every month or two. The deeper processes have been much stubborner and in our hands usually require treatment for many months. As has been observed by many, these cases are all especially liable to recurrences even when the patient's condition becomes robust.

We have a certain amount of money at our disposal to help worthy patients who can not follow out directions on account of lack of means. Those who have not homes where proper treatment may be carried out are referred to various institutions which have facilities for handling tuberculosis. Occasionally we meet a case in which class treatment is not successful. These are referred to the sanatoria; just as in all medical cases each patient must be treated as an individual and the treatment modified to suit his wants.

The paper referred to above[1] stated that the writer (G. S. D.) had been convinced of the value of tuberculin. As the result of further observation we wish to modify this statement. In the classes referred to tuberculin is used in all severer inflammations. As a rule the phlyctenular case does not need it. We believe that, as a rule, our patients do better when tuberculin is used. We employ now a bouillon filtrate obtained from Saranac and begin ordinarily with an initial dose of 0.0001 mg. It has seemed to us that our patients did better and suffered less harm on small and increasing doses rather than when given larger doses for the purpose of stirring up a slight local reaction. We have, however, seen in certain instances a marked improvement follow the diagnostic injection of old tuberculin, and in one instance, a case which had reached a standstill, the patient began to improve again after one or two large doses had been tried; but we have also often seen setbacks result from too large dosage, and therefore we do not believe in it as a routine procedure. We believe that the use of tuberculin should always be second to strict hygienic treatment even if the patient be in apparently good condition, for we know of the great good which may be accomplished in this way alone.

To conclude, allow us to report an interesting and severe case that resisted class treatment and tuberculin but yielded readily after the patient was sent to a sanatorium.

History.—M. M., a man, aged 26, was referred to us for treatment May 5, 1908. Following measles when he was 11 years old he had numerous attacks of ocular inflammation which seriously damaged his sight and kept him out of work. During several of these attacks he was treated as an inpatient in the Massachusetts Charitable Eye and Ear Infirmary. The attacks were so frequent that he was able to work only five months all told out of the last thirty-six.

Examination.—The patient had swollen lids, intense photophobia and lachrymation; both corneæ were covered with old scars and a well-marked pannus. There was no trachoma. V. o. u. = hand movements. It was necessary to lead the patient. General examination showed a poorly nourished man with the signs of an old pulmonary tuberculosis at the left apex. Cutaneous reaction positive. Temperature elevated. The patient was in a most despondent condition.

Course of Disease.—In conjunction with the Massachusetts General Hospital Tuberculosis Class we took charge of him and a strict antituberculous regimen was prescribed, with tuberculin injections and appropriate local treatment. The patient was helped financially, as, outside of what his wife could earn cooking, they had no means of support. Although we did everything in our power for him, his improvement was very slow, and at the end of ten weeks we decided to send him to the state sanatorium at Rutland, at which institution he was admitted July 15. Here his improvement was most rapid; the ocular inflammation had subsided at the end of five weeks, and his vision had increased to 10/200 o. u., as much as he had had for some years previously, according to his former hospital record. He left Rutland after seven weeks, having gained 24 pounds and feeling in better condition than ever before in his life. He resumed his work in a golf-ball factory and has not missed a day in the last six months. He reports occasionally for observation and is careful of his method of living.

7 Hereford St., Boston—54 West Main St., Westboro.

MEMORANDA

NEUROPATHIC KERATITIS AND SOME ALLIED CONDITIONS, WITH SPECIAL REFERENCE TO TREATMENT *

F. H. VERHOEFF, A.M., M.D.
Pathologist and Assistant Ophthalmic Surgeon, Massachusetts Charitable Eye and Ear Infirmary; Instructor in Ophthalmic Pathology, Harvard University
BOSTON

During the past four years I have given special attention to all cases of eye inflammation that I have had reason to believe were of neuropathic origin. As a result of my observations I have become convinced that lesions of the eye, due to disturbance of innervation, are too often incorrectly diagnosed and treated. Moreover, I feel sure that many cases of keratitis reported in the literature as unusual are really examples of neuropathic keratitis which do not happen to correspond with textbook descriptions. No doubt in some instances the real nature of a case of neuropathic keratitis is overlooked in the attempt to assign to it a bacterial origin.

The only neuropathic conditions of the eyeball due to disturbance of sensory nerves about which anything definite seems to be known are confined to the cornea, iris and conjunctiva. Of these only neuropathic keratitis is at all well recognized. The latter term is not here used synonymously with the so-called neuroparalytic keratitis as is sometimes done, but in the broad sense indicated by its derivation, that is, to designate any lesions of the cornea due to disturbance of its nerve supply.

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CONDITIONS EMBRACED IN NEUROPATHIC KERATITIS

The corneal conditions now generally accepted as neuropathic in origin are classified as herpes zoster corneæ, keratitis neuroparalytica, herpes febrilis corneæ and herpes corneæ neuralgicus. This classification, so far as it goes, is relatively satisfactory, if it is understood not to correspond strictly to differences in etiology. There are probably many, however, who still cling to the traumatic theory of neuroparalytic keratitis, but critical analysis made by Wilbrand and Saenger[1] should convince anyone that this is untenable. The simple fact, it seems to me, that neuroparalytic keratitis never presents any features that have not occurred also in the keratitis of herpes zoster is sufficient evidence that the two are produced in the same way.

In addition to these forms of neuropathic keratitis, several other corneal conditions have been described which are, perhaps, not generally accepted as neuropathic, but which I think should be included under this head. These are dendritic keratitis, superficial punctate keratitis, keratitis profunda, keratitis disciformis, and traumatic relapsing erosion of the cornea. My reason for holding this view is that the corneal changes considered characteristic of these conditions are not infrequently encountered in undoubted neuropathic keratitis, such as herpes zoster corneæ and herpes febrilis corneæ, and that typically they are associated with corneal hypesthesia and give the characteristic staining with fluorescin. Thus, dendritic keratitis may occur under the same conditions as herpes febrilis corneæ and is often associated with herpes labialis. The dendritic figure, moreover, may be combined with other features of neuropathic keratitis, such as superficial punctate spots and even vesicles. The malarial keratitis of Kipp[2] is undoubtedly identical with dendritic keratitis and is, therefore, not characteristic of malaria, except probably to the extent that neuropathic keratitis of malarial origin almost always assumes this form.

Superficial punctate keratitis, Fuchs[3] states, is allied to herpes febrilis corneæ, but he does not expressly state that it is neuropathic in origin. In the cases I have seen the staining with fluorescin has been characteristic and there has been hypesthesia of the cornea. It seems to me probable that the condition is really identical with herpes febrilis corneæ, all the lesions by chance taking the punctate form instead of only a few as is usually the case. It is possible, however, that cases due to bacteria may occur presenting this corneal picture. In fact, it is known that this actually occurs in leprosy, and Herbert[4] has described what he regards as bacilli in the epithelial cells in cases seen in Bombay. The bodies described by Herbert, however, may have been products of cell necrosis.

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[1] Wilbrand and Saenger: Die Neurologie des Auges, Wiesbaden, 1901, vol. ii.

[2] Kipp, C. I.: Keratitis from Malarial Fever, Tr. Am. Ophth. Soc., 1880, p. 91.

[3] Fuchs, E.: Lehrbuch der Augenheilkunde, Leipsic, 1907.

Keratitis profunda and keratitis disciformis Fuchs considers to be distinct conditions. The only constant difference between them, however, seems to be the presence of a gray border around the disc in the latter form. Fuchs holds the view that disciform keratitis is due to some unknown micro-organism which gains entrance through the abrasions resulting from herpes or trauma. Von Hippel[5] does not discuss the possibility of a neuropathic origin, but thinks that both conditions are due to changes or defects in the endothelium which allow the aqueous humor to infiltrate the cornea, and suggests that if Fuchs' view in regard to infection is correct the infection may come from within. He does not believe that the two conditions can always be sharply differentiated from each other. Peters[6] maintains that keratitis disciformis is due to a disturbed innervation leading to edema and necrosis and that the endothelium is secondarily affected. Meller[7] made a microscopic examination of an eye showing the condition in an advanced stage and concluded that Fuchs' explanation was correct. It does not seem to me, however, that this conclusion was warranted by his findings. The facts that both keratitis profunda and keratitis disciformis occur most commonly after herpes zoster ophthalmicus or trauma, that both may be associated with corneal hypesthesia and show outlying superficial punctate spots, impel me to believe that both conditions are essentially neuropathic, the minor differences between them being dependent on differences in the intensity of the nerve irritation.

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[4] Herbert, H.: Ophthal. Rev., 1901, p. 339.

[5] Hippel, E. von: Die Ergebnisse meiner Fluoresceinmethode zum Nachweis von Erkrankungen des Hornhautendothels, Arch. f. Ophth. (Graefe's), 1902, liv, 509.

[6] Peters: Ueber traumatic Hornhauterkrankungen und ihre Beziehungen zum Herpes corneæ, Arch. f. Ophth. (Graefe's), 1904, lvii, 93.

[7] Meller, J.: Die histologischen Veränderungen des Auges bei der Keratitis disciformis, Klin. Monatsbl. f. Augenh., 1905, li, 335.

The condition known as traumatic corneal erosion, which is undoubtedly an exaggerated form of traumatic keratalgia, has been explained by assuming that the epithelium of the cornea, owing to the injury, does not regenerate normally, and is easily brushed off by the lids, especially after the latter have adhered to it during the night. Wickerkiewicz[8] and others, however, have explained it as due to injury of the corneal nerves, and attribute its absence after more severe injuries to the fact that in the latter case the nerves are actually broken. This view seems to me the correct one, because the keratitis produced may simulate almost any of the forms of neuropathic keratitis just described, and there is hypesthesia of the cornea as well as the characteristic staining with fluorescin. Recently Cuperus[9] has brought forward the view, based on the fluorescin test, that the cornea is not normal between the attacks, which I can confirm by my own observations. I am sure, too, that if this test is always carefully applied, few cases of traumatic keratalgia will be met in which corneal lesions are not seen. The neuropathic theory of relapsing traumatic erosion of the cornea accords also with the fact that a special kind of injury is necessary, such, for instance, as that due to the snap of a twig. The injury to the nerves thus produced, however, does not always lead to actual erosions, but, as already indicated, may produce a condition indistinguishable from keratitis profunda. This condition I have seen in almost all cases of traumatic neuropathic keratitis in which the injury did not act directly upon the cornea but through the lid.

It might be expected that in neuropathic keratitis in which the epithelium is almost always altered if not actually lost for a considerable period of time, infection would be a frequent occurrence. This, as a matter of fact, is not the case, for serious infection occurs in only a small percentage of the cases. Since, however, neuropathic keratitis is relatively common, the small number of infected cases constitute a large percentage of all cases of ulcerative keratitis. In fact, while I have no exact statistics bearing on this point, I believe that they constitute the largest percentage of such cases, exclusive of gonorrheal and phlyctenular disease. Some of the worst cases of ulcus serpens have a neuropathic basis, traumatic or otherwise. In these cases, unless seen at an early stage of the infection, it is difficult to determine the neuropathic element. According to Peters,[6] it is the neuropathic element to which ulcus serpens owes its peculiar characteristics.

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[8] Cited by Wilbrand and Saenger: Die Neurologie des Auges, ii, 84.

[9] Cuperus, N. J.: Ueber sogennante recidivirende Erosio corneæ. Arch. f. Augenh., 1908, lxi, 26.

PATHOLOGY OF NEUROPATHIC KERATITIS

Knowledge of the pathology of neuropathic keratitis is based almost entirely on postmortem examinations made in cases of herpes zoster and neuroparalytic keratitis. Few observations have been made in connection with the histology of the corneal lesions, on account, no doubt, of the difficulty in obtaining material at a sufficiently early stage and uncomplicated with lagophthalmic keratitis. The work of Head and Campbell[10] on herpes zoster, and the admirable exhaustive review of the whole subject by Wilbrand and Saenger,[1] have, I think, established the fact that neuropathic keratitis is due to an irritative lesion of the fifth nerve or Gasserian ganglion. Head and Campbell maintain that in zoster the most important lesion is in the ganglion, but Wilbrand and Saenger give good reasons for believing that the essential lesion is a neuritis. As to the peripheral changes in the skin and cornea, Head and Campbell do not give any definite explanation, but state that they do not assume the existence of trophic nerves. Wilbrand and Saenger, on the other hand, think that the peripheral lesions are produced in some obscure way by abnormal impulses from trophic nerves. The existence of trophic nerves, however, has never been demonstrated. I would suggest a theory which, while admitting the possibility, does not necessitate the assumption of trophic nerves, and which seems to explain more definitely the production of the peripheral lesions. It is a well-known physiologic fact that if a nerve of any kind is stimulated, impulses, as shown by the galvanometer, pass along it in both directions.[11] Assuming now that such impulses are produced by some lesion of the fifth nerve, it is self-evident that the efferent impulses must give rise to some changes, however, slight, at the nerve terminals. One of the simplest changes that could be imagined, and one that would seem to be physiologically possible, would be the production of an acid reaction. If these impulses were continued for a considerable period of time, sufficient acid or other irritating substance might be produced as to cause the lesions in both the skin and the cornea.

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[10] Head and Campbell: The Pathology of Herpes Zoster, Brain. 1900, p. 353.

This theory of neuropathic keratitis harmonizes perfectly with the recent theory of Fischer[12] in regard to the nature and cause of edema. In herpes of the cornea the occurrence of vesicles leaves no doubt that here is a local edema. This, it seems to me, establishes the fact that edema is not dependent essentially on changes in blood vessels—a fact that is not elsewhere easy of demonstration. It might be urged that the vesicles on the cornea were due to a general edema dependent in some way on the vessels around the cornea or within the globe, were it not that the vesicles often occur and remain definitely localized. According to Fischer, the cause of edema in general is an increased affinity of the tissue colloids for water. This increased affinity may be brought about by a variety of chemical changes, the most important of which is probably the occurrence of acid. It is apparent at once how satisfactorily this theory may be applied in the explanation of the lesions of neuropathic keratitis. Thus the irritative lesion, acting on the fifth nerve or Gasserian ganglion, sends impulses to the cornea which produce an acid reaction or other chemical change at the nerve terminals. This in turn acts on the colloids of the corneal lamellæ or epithelium, causing them to become edematous. The edema affecting the epithelium from behind produces vesicles, erosions, or less marked changes. To explain the denser and, to a greater or less degree, permanent opacities which occur in cases of long duration, it is only necessary to assume further that proliferative changes take place. These could be produced by the action of the irritant on the corneal corpuscles leading either directly to proliferation or to necrosis followed by the process of repair.

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[11] There is evidence also that even under normal conditions efferent impulses, called antidromic impulses, may pass along sensory nerves. Without regard to this possibility it seems probable that all nerves, both afferent and efferent, may in a sense act as trophic nerves, because it is almost self-evident that there must be some reciprocal relationship between the vital activities of the tissue cells and of the nerve filaments with which they are so closely related anatomically.

[12] Fischer, M. H.: The Nature and Cause of Edema, THE JOURNAL A. M. A., Sept. 5, 1908, li, 830.

The variations in the corneal changes in neuropathic keratitis could be readily explained by variations in the intensity of nerve irritation and in the number and distribution of the nerve fibrils involved. The sharp definition of many of the lesions, notably the punctate spots and dendritic figured, may perhaps be simply an expression of the fact that the fibrils in the nerve trunks are collected into bundles. Wilbrand and Saenger have offered an ingenious explanation of the variations that occur in different cases in regard to pain and disturbance of corneal sensibility. This is, briefly, that these variations are dependent on whether the nerve lesion produces impulses in one or both directions, and on the extent to which it blocks off impulses in one direction or the other. It is obvious that this explanation is applicable whether or not the existence of trophic nerves is admitted.

ETIOLOGY OF NEUROPATHIC KERATITIS

As just explained, neuropathic keratitis is almost certainly due to an irritative lesion of the fifth nerve or its ganglion. The character of this lesion otherwise seems to be unimportant, so that similar corneal changes may be produced by various causes, such as trauma, tumors, meningitis, tubercle, syphilis, hemorrhage, or to primary neuritis. In a particular case, therefore, it may be difficult or impossible to determine the etiologic factor. Head and Campbell[10] assert that herpes zoster is a specific infectious disease analogous to acute poliomyelitis. This may be true of the majority of cases, yet a condition indistinguishable from zoster may occur in the course of other infections, such as influenza, or may be produced by trauma or by the action of poisons, especially arsenic. Some observers have even asserted that an atypical form of zoster may be produced by hysteria. In certain cases of herpes corneæ neuralgicus, periodical eruptions of vesicles on the cornea have coincided with the menstrual periods.

Probably the largest number of cases of neuropathic keratitis come under the designation of herpes febrilis corneæ. In most of these there is a history of a recent attack of coryza, bronchitis, pneumonia, malaria, or gastrointestinal disturbance, and most often there is herpes facialis or labialis. Cases are met, however, in which no etiologic factor can be determined, so that the distinction between them and herpes zoster corneæ without skin involvement, the so-called *forme fruste,* is obscured. It is probably also that the conditions which ordinarily produce herpes febrilis may exceptionally give rise to a skin eruption identical with that of zoster.

As already stated, trauma of a certain kind acting on the cornea may produce almost any of the corneal lesions of neuropathic keratitis. This is true also of trauma acting on the nerve trunk or Gasserian ganglion from any cause, such, for instance, as fracture of the skull. The indefiniteness, from an etiologic standpoint, of the distinctions between the various classes of neuropathic keratitis is exemplified also by the so-called neuroparalytic keratitis. This term, as commonly employed, seems to be incapable of an exact definition, and is used in an entirely arbitrary manner. It can not be defined on the basis of etiology, since the same etiologic factor, trauma, for instance, may at one time produce neuroparalytic keratitis and at another a condition indistinguishable from herpes zoster. Probably it is most often applied to cases which can not be included under herpes zoster or herpes febrilis. It is used especially to denote the keratitis due to extirpation of the Gasserian ganglion, syphilis, etc., and that due to general nervous disease, such as tabes.

DIAGNOSIS OF NEUROPATHIC KERATITIS

As already indicated, in the majority of cases that come to the attention of the ophthalmologist, the remote causes underlying the nerve lesion seem to be of relatively little importance and, in fact, not clearly understood. On the other hand, in cases of brain tumor, fractured skull, tubercle, meningitis, tabes, etc., the eye condition may be only an unimportant symptom the remote cause of which is at once sufficiently obvious. In any case the possibility of poisoning, especially with arsenic or lead, should not be lost sight of.

In making the diagnosis of the keratitis itself the important factors to be considered are the sensibility of the cornea, the appearance of the corneal lesions, especially after staining with fluorescin, the injection of the eye, the intraocular tension, the size of the pupil, the subjective symptoms, and the occurrence of herpetic skin eruptions. The testing of the relative sensibility of the cornea requires care in order that the results may be of any value. When the sensibility is completely abolished the condition is, of course, easily determined, but this occurs in very few cases. I have found a piece of thread, as recommended by Fuchs,[3] the most satisfactory test object. More precise methods have been devised, such as blowing a stream of air on the cornea, but in my opinion these are too delicate to be of any practical value. For clinical purposes it is also not practical to differentiate the senses of touch, pressure, temperature and location. I am sure that any change in the sensibility of the cornea to which it is safe to attach any importance can be determined by the thread test if it can be determined at all. The test should be made with reference both to the lid reflex and the sensations of the patient. The former is the only one which can in many cases be relied on, especially in neurasthenic individuals. Not infrequently the lid reflex may be elicited without the patient being fully aware that the cornea has been touched at all. It is best determined by having the patient keep his eyes open without assistance. Unfortunately, many patients as soon as their eyes are approached put their eyelids into a more or less spastic condition which inhibits the lid reflex. In some cases it is necessary to raise the eyelid with the thumb. This is not so unsatisfactory as might be thought, because here the reflex may be both felt and seen by the observer. The cut end of the thread should not be punched directly into the cornea, but rubbed over the surface, the pressure being increased as found necessary. The same path should never be repeatedly traversed, as this may render the epithelium more sensitive. The unaffected eye should, of course, always be tested as a control.

The sensations of the patient may be ascertained at the same time the test for the lid reflex is made. It is always a surprise to the patient to find that the affected eye is less sensitive than the other, and he may answer incorrectly for psychologic reasons. This fact, however, obviously makes the test more reliable when hypesthesia is found. Many patients are influenced by seeing or feeling the movements of the hand making the test, so that these should be masked as far as possible. I always make several preliminary movements before actually touching the cornea, and not infrequently, without any intention to deceive, the patient states that a sensation is felt. This may happen when the cornea is completely anesthetic, and is probably due to the irritable condition of the cortical centers produced by the constant impulses from the nerve lesion. Too much reliance should not be placed on diminution of sensibility over infiltrated areas, as this might occur in other conditions. Various portions of the cornea should always be tested, since the sensibility may be more impaired in some than in others. The periphery of the cornea is normally less sensitive than the center.

The importance of testing the sensibility of the cornea for the diagnosis of neuropathic keratitis is exceedingly great, for while it may be impaired in other conditions, notably in glaucoma and late stages of syphilitic interstitial keratitis, these as a rule are easily distinguished from neuropathic keratitis. In the early stages of interstitial keratitis I have found the corneal sensibility normal or increased, even over the infiltrated areas. There is considerable variation in the sensibility of the cornea among normal individuals; in old age it is apt to be markedly diminished, but the unaffected eye usually offers a satisfactory control here. Cases of neuropathic keratitis are said to occur in which the sensibility of the cornea is normal or increased, but these are too infrequent to be of much importance.

The most characteristic corneal lesions in neuropathic keratitis consist of erosions, vesicles, filaments (filamentous keratitis), superficial punctate spots, and infiltrates or ulcers of more or less dendritic form, occurring singly or in different combinations. Peters has pointed out that the epithelium may be loosened and movable over the affected areas. In cases of long standing the cornea may show permanent opacities and vascularization. The new vessels may be either deep, superficial or in a pannus formation, and are usually few but of large caliber. They are rarely so deeply seated as those in the interstitial keratitis of syphilis. In many cases the corneal lesions are readily seen, but in others they may be overlooked if the fluorescin test is not used. Simple as this test is, it is seldom properly applied and its full value is not generally appreciated. Usually after instilling a drop of the solution the intense stain, which immediately occurs when erosions are present, is alone looked for, whereas this is relatively unimportant, since erosions as a rule may be seen unstained. More important is the light green staining, which is either superficial or relatively deeply seated, and which is not due to actual erosions. To bring this out the fluorescin solution should be allowed to act one or two minutes, and if there is profuse lachrymation a second drop should be instilled. The conjunctival sac should then be washed practically free from fluorescin with boric acid solution. This is important because the film of fluorescin solution over the surface of the cornea may make opacities of any kind, or facets, appear slightly green, or, on the other hand, may mask the areas in the cornea which are actually stained.

Staining of the cornea with fluorescin, exclusive of erosions, occurs in conditions other than neuropathic, but these are usually easily recognized. It occurs in glaucoma and in lagophthalmic keratitis.[13] Deeply seated staining, according to von Hippel,[5] is always due to injury of the corneal endothelium. In regard to glaucoma I can confirm his observations, for I have found the endothelium altered or absent in a number of eyes enucleated for this condition. In one of these cases I observed deeply seated fluorescin staining forty-eight hours before the enucleation. It does not necessarily follow, however, that the injury to the endothelium was the cause of the staining, because both may have been due to changes within the cornea associated with edema. I have failed to obtain any staining in tuberculous interstitial keratitis except in cases in which the epithelium was affected by an infiltrate lying just beneath it. According to von Hippel, there is a limited class of cases of interstitial keratitis due to congenital syphilis in which the infiltrates are chiefly central, which show deep staining with fluorescin. This staining has also been seen in cases of sympathetic ophthalmitis, in which

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[13] Fluorescin staining similar to that seen in lagophthalmic keratitis may often be obtained in the normal eyes of rabbits or guinea-pigs, being due no doubt to the infrequency of winking in these animals.

I have myself observed it. I have seen it also in certain cases of iritis which seemed to be undoubtedly syphilitic. Von Hippel apparently did not consider the possibility of a neuropathic basis in any of his cases. Considering the profound changes that may occur in the epithelium in neuropathic keratitis, it would not seem strange if the endothelium should also sometimes suffer. Possibly some of the cases of iritis described by von Hippel were similar to the cases of neuropathic iritis considered below.

The injection of the eye in neuropathic keratitis is almost always entirely conjunctival, even when there is iritis. The amount of injection seems to bear no relation to the degree or character of the corneal involvement. Thus a marked conjunctival injection associated with chemosis may occur while the cornea shows a few lesions brought out only by the fluorescin test. This condition is apt to be mistaken for an infectious conjunctivitis. On the other hand, the cornea may be cloudy throughout and almost completely denuded of epithelium without any ocular injection. When either of these conditions occurs it obviously assists in the diagnosis. There does seem to be some relation, however, between the amount of injection and the amount of pain, although this is not as close as might be expected. In most cases the injection is no doubt due to a reflex vasomotor dilatation arising from the central irritation produced by the nerve lesion. In the marked cases it does not seem possible that this could be the sole cause, but that it is here due largely to the direct irritation of the nerves supplying the conjunctiva. In this way also may be explained the edema of the eyelids which is sometimes associated with neuropathic keratitis. In cases of infection the injection is increased, but often less than would be expected.

The pupil in neuropathic keratitis is usually somewhat smaller than in the normal eye. In exceptional cases, however, there has been mydriasis due to an associated paralysis of the third nerve. The intraocular tension is apt to be subnormal, but seldom is it decidedly so. In rare cases glaucoma has ensued.

The subjective symptoms, like the ocular injection, bear no constant relation to the amount of corneal involvement. Pain of a neuralgic character, referred to the eye or neighboring parts, may be extremely severe or entirely absent. Often it is present at the onset only, but it may persist throughout the attack. The feeling of foreign body irritation is common, probably due to the presence of erosions. It may be absent, however, with the greater part of the cornea eroded. Not infrequently all that is complained of is dimness of sight. In such cases the corneal infiltrates may be mistaken for old scars if the fluorescin test is not applied.

The presence of an herpetic eruption on the face should, of course, always be looked for. When the eruption is extensive, as in well-marked cases of herpes zoster, it is too obvious to escape notice, but it may be mistaken for erysipelas. Sometimes all that may be found are one or two vesicles on the side of the nose or high up on the forehead. If herpes of the lips is not present at the time of the examination, inquiry will often elicit the fact of the recent occurrence of a "cold sore."

TREATMENT OF NEUROPATHIC KERATITIS

The local measures usually employed in the treatment of these patients have been the use of the bandage, atropin, ointments, and in severe cases the application of irritating or caustic agents, such as tincture of iodin or the actual cautery. None of these, however, can be said to be entirely satisfactory, although in mild cases an occlusive bandage may be alone sufficient. The use of the actual cautery has recently been strongly recommended by Amman[14] for herpes febrilis corneæ. Cauterization may no doubt in some cases put an end to the process, but only at the sacrifice of more or less corneal tissue, so that the resulting opacity is probably as great, if not greater, than if the case had remained untreated.

Within the past two years I have adopted a method of treatment, founded on what I believed to be a rational basis, which has proved uniformly successful. It occurred to me that since neuropathic keratitis was due to an irritative lesion of a sensory nerve, recovery ought to follow if the nerve terminals were anesthetized by means of a local anesthetic. Even if permanent anesthesia proved impracticable, it seemed that treatment along this line should lead to improvement in the condition. As an anesthetic for this purpose I selected holocain, for several apparently unimportant reasons, one of which was that it was slightly antiseptic and that its solutions remained sterile. My results were so satisfactory that I have not tried any of the other anesthetics, although it is possible that some of the latter might prove still more efficient. The most important reason, however, for continuing with holocain was that by adopting a single method of treatment in a series of cases, which were extremely variable anyway, it would be possible to arrive at more accurate conclusions as to its value than if the treatment was continually being changed.

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[14] Amman, E.: Neues Heilungsverfahren beim Herpes Corneæ febrilis, Arch. f. Augenh., 1908, lxi, 194.

Owing to its toxicity it did not seem advisable to use holocain in oily solution or in the form of an ointment, and from the beginning I have used a 1 per cent. aqueous solution. After some experimentation in regard to the frequency of the instillations, I concluded that intervals of no more than two hours gave the best results, and that it was no advantage to make them as short as one hour. I also concluded that a bandage was essential, and of this I feel certain. The bandage that has proved most acceptable to the patients has been one of the well-known celluloid eye-shades with an eye-pad placed beneath. This has the advantage of being cooler, easier to remove for the frequent instillations, and of being less unsightly than an ordinary bandage. The elastic cord should be adjusted to give just sufficient pressure to keep the eyelids closed and no more. In one very marked case, in which only a rim of epithelium remained at the periphery of the cornea, I obtained more rapid improvement by bandaging both eyes. Patients in such cases as this and patients badly infected at the outset are the only ones I have not treated as out-patients. In all cases the bandage and instillations were omitted during the night.

As a rule a mydriatic was not prescribed unless there was a well-marked iritis. If the pupillary reaction was not quite normal it was generally sufficient to instill a drop of atropin solution at the time of each visit. At the suggestion of Dr. P. S. Smith, I have sometimes added scopolamin hydrobromate in the proportion of 1/300 to the holocain solution with satisfactory results, but I can not say that a small amount of atropin would not prove equally satisfactory.

I have now treated over one hundred patients by this method, and have yet to see a patient who did not improve from the time the treatment was begun. In some cases the cornea failed to stain with fluorescin within a week; in others more or less staining was obtained for a number of weeks. Usually this involved only one or two punctate spots which could easily have been overlooked if not carefully searched for. The treatment was continued until not the slightest staining could be obtained. The bandage was then dispensed with unless, as sometimes happened, the patient desired to wear it longer.

Local treatment of the eye could not, of course, have any influence on the source of the nerve irritation so that it might be expected that when it was discontinued the keratitis would recur. This did, in fact, sometimes happen, but in some cases, which no doubt would have run a short course anyway, the keratitis did not recur even when the local treatment was discontinued within one week. No doubt in such cases the nerve lesion had within this time undergone partial or complete retrogression. In a number of cases recurrences took place repeatedly so that it became advisable to try some general treatment.

It suggested itself to me that since arsenic seemed to have a special affinity for nerve tissue, as indicated by the well-known fact that it may produce an herpetic skin eruption, it might act in a specific manner on the nerve lesion in neuropathic keratitis. Whether or not this is really the case, my results following the administration of arsenic have been most satisfactory. In cases which over a period of several months had recurred whenever the local treatment was discontinued, the lesions, after several weeks' treatment with arsenic, became permanently healed. Since it is impossible at the outset to determine whether or not a case will assume this chronic type, to save time I now give arsenic in all non-traumatic cases in which some other general treatment is not indicated. My custom has been to order for adults two drops of Fowler's solution three times daily, to be increased one drop each day until ten drops is reached, at which dose it is continued for a month or longer if necessary. The patients are, of course, warned to note the symptoms of arsenic poisoning, such as gastritis and enteritis.

The cases of neuropathic keratitis in which the treatment with arsenic was not indicated have been very few. In two cases in which lead-poisoning was suspected the use of potassium iodid was followed by prompt recovery. This was also used at first in a few patients with syphilitic histories, but with apparently no effect, whereas the arsenic treatment was successful. In several patients with histories of repeated malarial attacks, but with no active symptoms of malaria, arsenic was used instead of quinin with satisfactory results.

The use of arsenic in neuropathic conditions is, of course, not entirely new, since it was long ago advocated for herpes zoster of the skin. Its beneficial action here is not so obvious, because it is comparatively slow in manifesting its effect. If administered early, however, it might be expected to prevent the disease from becoming unusually prolonged. Then, too, almost thirty years ago Kipp[2] stated that he had given arsenic, in medium doses, in many cases of malarial keratitis with very beneficial results.

What has been said so far in regard to treatment has applied especially to neuropathic cases uncomplicated by infection. It is noteworthy, however, that I have not had infection occur in a single case after the holocain treatment was begun. On the other hand, I have seen several patients with infected ulcers and hypopyon recover promptly under this treatment alone; in fact, I have found this treatment advantageous in all forms of ulcerative keratitis in which it has been tried, whether on an evident neuropathic basis or not. In cases of ulcus serpens additional measures have been required, such as cauterization and the Saemisch operation. The holocain treatment has proved most efficient also in cases of keratitis from acne rosaceæ.

While I have no doubt of the value of holocain in the treatment of neuropathic keratitis, when used in the manner described, it is possible that its therapeutic effect may be dependent, in part at least, on some principle other than that which led me to employ it. Certainly the anesthesia produced in a normal cornea by one instillation of holocain does not persist two hours, though it is possible that its effect on nerves constantly irritated may be more lasting. The possibility must be considered that its action may be directly on the tissues, especially on the epithelium. As is well known, solutions of any of the local anesthetics, when repeatedly instilled into the eye at short intervals, produce decided pathologic changes in the epithelium, leading even to necrosis.[15] It may be, therefore, that holocain in a 1 per cent. solution, used every two hours, acts as a stimulant to the tissues. I am in a position to state positively, at any rate, that it does not harm the epithelium even when used in this way for several months, and that erosions of the cornea are rapidly repaired under its use.

NEUROPATHIC IRITIS

In cases of neuropathic keratitis the iris is not infrequently involved. Usually the iritis is of a mild type and the synechiæ give way readily, but in exceptional cases it may predominate over the keratitis. It is known also that iritis may occur in herpes zoster ophthalmicus without involvement of the cornea. There can be no doubt, therefore, that iritis may be produced by the same factors that give rise to neuropathic keratitis. This naturally raises the question whether or not neuropathic iritis may occur unassociated with either keratitis or skin eruptions, that is, without any obvious indication of its neuropathic origin.

I have seen several cases in which I believed that this condition might have existed. Of these I have records of four. In three of these the diagnosis was based chiefly on well-marked hypesthesia of the cornea. In one case, although the cornea remained practically clear, at the end of a week the fluorescin test showed an area of faint green staining apparently just beneath the epithelium. In two of the cases slight fluorescin staining was obtained when first seen. In two of the three cases the onset was with intense supraorbital neuralgia. The fourth case was more suggestive still. In this there had been an attack of superficial punctate keratitis six weeks previously which had responded promptly to the holocain treatment. After an interval of about four weeks a herpetic eruption appeared on the lip on the same side. Two weeks later the patient came to see me, complaining of the most intense neuralgia which had come on abruptly in and about the eye. There was much photophobia and marked conjunctival and ciliary injection. The iris was discolored and showed synechiæ, which, however, readily gave way under atropin. There was no hypesthesia of the cornea and the fluorescin test was negative, although the punctate opacities from the previous keratitis were still visible.

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[15] The relative toxicity of various local anesthetics has recently been studied by Guillery (Die Bedeutung der Anästhetica für die Behandlung der durch Aetzgifte entstandenen Hornhauttrübung, Arch. f. Augenh., 1908, lxi, 38). Under the conditions of his experiments holocain, 2 per cent. solution, seemed the most toxic of all. His results, however, are of limited significance from a therapeutic standpoint, since different results might well have been obtained by varying the concentrations of the solutions and the frequency of their instillations. Other factors, also, such as duration and rapidity of action, here require consideration.

While these cases may show that neuropathic keratitis occurs without keratitis, they give no indication of its frequency. It is obvious that should cases occur without hypesthesia and without the circumstantial evidence afforded by the last case, the correct diagnosis would be difficult if not impossible to make. They would naturally be classed as cases of rheumatic iritis, which brings up the question whether many cases so called are not neuropathic in origin. In regard to this I have not sufficient evidence to venture an opinion. In view, however of my satisfactory results in the treatment of patients in intractable cases of neuropathic keratitis with arsenic, it would seem well to try this treatment in any case of iritis of unknown etiology which failed to respond to the usual methods.

NEUROPATHIC CONJUNCTIVITIS

As is well known, in neuralgia of the fifth nerve from any cause there may be hyperemia of the conjunctiva. This is clearly analogous to the conjunctival hyperemia occurring in association with neuropathic keratitis, and no doubt due to the same cause. It may be so marked as to be mistaken for an infectious conjunctivitis, but the neuralgic pain and the lack of purulent secretion usually make the diagnosis easy. In some cases there is hypesthesia of the cornea and conjunctiva. Not much reliance, however, can be placed on a test of the sensibility of the conjunctiva, because normally it is not very sensitive. Other forms of conjunctival hyperemia, such as that due to reflex irritation from the nasopharynx, will not be considered here, since they are not dependent on actual lesions of the fifth nerve. The hypesthesia of the cornea in cases of xerosis suggests a neuropathic basis for this rare condition. This is supported by a pathologic examination made by Circione.[16]

Within the past two years I have come to recognize another condition of the conjunctiva as neuropathic, to which I desire to call especial attention. It is not uncommon and is usually designated by the broad term "episcleritis," under cover of which our ignorance in regard to several ocular affections has frequently been hidden. The condition manifests itself as a localized area of congestion in the center of which the conjunctiva is swollen into a more or less definite nodule. There is also a variable amount of general conjunctival hyperemia. The epithelium over the nodule may be apparently normal, but at some stage it will usually stain with fluorescin. In some cases the surface of the nodule becomes eroded or ulcerated. The nodule may be situated anywhere on the bulbar conjunctiva, not infrequently exactly at the limbus. When in the latter situation the contiguous corneal tissue is apt to be hypesthetic, show localized cloudiness, and stain with fluorescin as in neuropathic keratitis. I have never recognized the condition on the palpebral conjunctiva. The duration is variable, generally from one to six weeks.

My evidence that this is a neuropathic condition, usually analogous to herpes febrilis corneæ, can be presented, perhaps, most briefly by stating the essential features upon which I based the diagnosis in sixteen consecutive cases seen during my last service of four months at the infirmary.

CASES OF HERPES OF BULBAR CONJUNCTIVA.

One case: neuralgia and corneal hypesthesia.

One case: corneal hypesthesia, neuropathic keratitis, edema of eyelids, coryza.

One case: herpes labialis, coryza.

One case: neuropathic keratitis in other eye, lead colic.

Three cases: corneal hypesthesia.

Two cases: coryza.

One case: corneal hypesthesia, herpes facialis.

One case: neuralgia, corneal hypesthesia, marginal keratitis.

One case: herpes zoster ophthalmicus, marginal keratitis and hypesthesia.

One case: dendritic keratitis, bronchitis.

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[16] Cited by Wilbrand and Saenger: Die Neurologie des Auges, ii, 51.

One case: marginal keratitis punctata, herpes labialis, coryza.

One case: marginal keratitis, and hypesthesia, bronchitis, herpes labialis.

One case: characteristic conjunctival lesion alone.

The case in the above list in which there was neuropathic keratitis in the other eye, it seems to me, was especially significant. The patient had slept in a freshly-painted room for three weeks and during the third week was taken with severe "cramps in the stomach," apparently lead colic. About one week later when seen by me the left eye presented typical neuropathic keratitis with hyperesthesia, while the right eye showed the conjunctival condition described, situated a short distance from the limbus. Both conditions practically disappeared in three weeks under the administration of potassium iodid.

In the list of cases, by "marginal keratitis" is meant the keratitis localized about the conjunctival nodule at the limbus. I have never seen this extend far into the cornea, probably because of the fact that the cornea and conjunctiva are supplied chiefly by different nerves, the cornea by the ciliary, and the conjunctiva largely by the supraorbital and infraorbital nerves. The conjunctival nerves, however, partly supply also the periphery of the cornea,[17] thus accounting for the extension of the conjunctival lesion into the latter.

As already stated, this condition of herpes of the bulbar conjunctiva is usually diagnosed as "episcleritis" and therefore not clearly differentiated from tuberculous scleritis or sclerokeratitis. It may also be mistaken for a large phlyctenule, especially when ulcerated, and probably it will not always be possible to avoid this error. I have always regarded with suspicion the alleged large solitary phlyctenules situated some distance from the limbus, and I am now inclined to believe that they are invariably of neuropathic origin.

When occurring in well-marked cases of herpes zoster ophthalmicus the neuropathic origin of the condition under consideration is, of course, evident. In this connection it is interesting that Wilbrand and Saenger[1] state that a "true episcleritis" may occur in herpes zoster. Judging by the description, the condition referred to would seem to be identical with that observed by me.

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[17] This possibly explains why a ring of epithelium is left on the cornea in severe cases of neuropathic keratitis. It suggests also a neuropathic origin for certain marginal ulcers.

In the treatment of herpes of the bulbar conjunctiva the same measures are evidently indicated as in neuropathic keratitis. Practically, however, I have found that treatment is seldom required except when the cornea is involved. Where the conjunctiva is alone affected and the subjective symptoms are negligible, it seems unnecessary to put the patient to the inconvenience of wearing a bandage, since there is no danger of serious infection and no scarring is to be feared as in the case of keratitis.

233 Charles Street.

MEMORANDA

LACHRYMAL GLAND TUMORS *

L. D. BROSE, M.D.
EVANSVILLE, IND.

A knowledge of the anatomy of the lachrymal gland is of prime importance to one who undertakes an operation for its removal, and therefore I offer a brief description of it.

ANATOMY OF THE LACHRYMAL GLAND

In character it belongs to the acinous gland and occupies a place in the upper outer and anterior portion of the orbital cavity, being lodged in what is termed the lachrymal fossa on the under surface of the orbital plate of the frontal bone. While in close apposition with the overlying periosteal covering at this point, it is, nevertheless, but loosely connected therewith. Through the expanded insertion of the elevator muscle of the upper lid the gland is divided into a superior, more compact portion of grayish-red color, and an inferior or accessory portion of looser texture and in close relation with the superior conjunctival fornix. Investing the gland is a fibrous capsule, which has numerous connections with the lateral anchoring fibrous orbital supports, extending between the superior rectus muscle and the outer orbital wall, and with the supraorbital periosteum through the suspensory glandular ligament. The inferior surface of the gland is concave and inclined downward and inward, being so placed as to give a transverse measurement according to Schwalbe[1] of from 20 to 25 mm., while the antero-posterior measurement is but 12 to 14 mm.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

[1] Schwalbe: Lehrbuch der Anatomie des Auges.

REPORT OF CASE

The following case is not only interesting but serves to bring out the symptomatology, diagnosis and treatment of tumor formations involving this gland:

History.—M. J. K., a well-developed, robust, married man, aged 45, a river pilot by occupation, consulted me Oct. 23, 1907, because of a growing hardness over the right eyeball. He first became aware of it three to four years before the consultation, since which time it had slowly increased in size, causing forward displacement of the eyeball with drooping of the upper lid. It had never been painful, but the eye when exposed to the inclement weather reddened and secreted tears.

Examination.—The cutaneous blood vessels were not unduly prominent, but at a glance marked ptosis was seen with forward, downward and inward displacement of the eye. Double vision did not exist and the patient denied specific infection. The left gland was not involved, neither had the patient glandular enlargement elsewhere in the body. On palpation a nonpainful enlargement of the right lachrymal gland was detected through the drooping lid; it was of firm texture, smooth of surface and in size that of a small English walnut. The growth was semi-movable, without intimate connection with the overlying bony wall, and when strongly pressed on, partially receded within the orbit, with corresponding increase in the forward and downward displacement of the eye. The patient did not complain of loss of vision. With the left eye he saw 15/xv and with the right eye 15/lx improved to 15/xx with — 1.00 cy., ax. 60. The fundus was without detectable lesion.

A diagnosis of probable adenoma of the lachrymal gland was made, and the patient was given iodid of potash three times daily, with the suggestion that he make arrangements to lay off from work and enter a hospital to undergo an operation for removal of the growth.

Operation.—On Feb. 14, 1908, the patient was operated on at the U. S. Marine Hospital under ether anesthesia, Dr. J. B. Stoner, surgeon in charge at this port, kindly assisting. After the eyebrow had been shaved and the field of operation sterilized with sublimate solution, an incision was begun over the external angular process and carried along the bony orbital rim inward to about one-third of the length of the upper lid. The convexity of the cut was directed upward and included the skin, subcutaneous tissue and orbicularis palpebrarum muscle. With retractors the lips of the wound were held apart, and next the tarso-orbital fascia carefully divided on a line corresponding with the skin incision and reflected downward. The growth appeared in the wound after this incision and was firmly grasped, with fixation forceps and dissected out with blunt, curved scissors. The adhesions were most extensive along the mesial and posterior inner part of the gland. After the hemorrhage had been controlled by gauze packing the wound was closed by four interrupted sutures and a sterile dressing applied.

Postoperative History.—Double vision was not encountered after the operation, but there followed some suppuration which after a time subsided with permanent closure of the wound, and up to the date of writing there has been no recurrence of the growth. The result of the operation has been marked recession of the eyeball and the ptosis, with retention of vision as before the time of operation. The growth was hardened in a solution of formaldehyd and a portion sent to Dr. H. R. Alburger, pathologist in the Indiana University, to whose kindness I am indebted for the following microscopic finding:

Microscopic Examination.—Section consists of a glandular mass made up of a stroma of connective tissue which is largely hyaline in character. Held in the meshes of this are found a number of gland tubules lined with columnar epithelium. In the main these tubules are regular and lined with a single layer of epithelium, but in several areas there is a lack of regularity of the tubes and the epithelium is in several layers and even growing in irregular masses without any distinct limiting membrane and with cells which show mitosis. Some of the more typical tubes contain a mass of structureless material, resembling colloid. Others contain mucus.

Diagnosis.—Adenoma of lachrymal gland undergoing malignant change.

CLASSIFICATION OF TUMORS OF THE LACHRYMAL GLAND

Tumors of the lachrymal gland may be primary in nature or secondary. They may be benign or malignant, and either of solid or cystic character, and may involve one or both glands at the same time. Of the reported cases by far the greatest number have been benign in nature, or at least their removal has not been followed by recurrence. Among the reported malignant growths we find the various sarcomas, chloroma and the different carcinomas. The pathologic histology of tumors involving these glands has led to considerable controversy and widely different conclusions. According to O. Becker and Rudolph Schirmer,[2] most of these tumors are properly classed as adenomas or adenoid formations, while Koester[3] and others classify them as cylindromas. "Endothelioma" is a better designation, since if by "cylindroma" is not meant a tumor springing from the endothelial lining of the lymph spaces there must remain doubt as to just what writers do imply by the designation. I can see no reason for believing that new formations in this gland prove an exception to those found in other acinous glands, such as the pancreas and salivary glands, to which, according to Schenk,[4] the lachrymal gland is histologically closely allied. I would in part reconcile the seeming contradictory views held by these investigators by the fact that tumors of the lachrymal gland, comparatively speaking, are of rare occurrence, and no one observer has met with a sufficient number of cases to establish therefrom a generally accepted basis of classification. A review of the histology of the benign tumors placed on record discloses that they have been of mixed character for the greater part, and this corresponds to what Weichselbaum[5] states concerning those found in the salivary glands. It is my belief that as a rule all the tissues entering into the formation of the lachrymal gland are involved in the growths occurring therein, with the preponderance of overgrowth in those elements of mesoblastic origin, and hence I would designate them according to the prevailing type of tissue, fibroma, adenoma, adenofibroma, or adenoendotheliofibroma. The designation "endothelioma" may properly be combined as in the foregoing when columns of endothelial cells are seen forming a network and retaining in places a lumen. When this arrangement is lacking the tumor, if histologically of endotheliomatous cell formation, is to be classed as sarcoma. In both the benign and malignant growths one may find, in addition to the epithelial and interacinous connective tissue proliferation, cartilage, bone, hyaline and colloid degeneration.

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[2] Schirmer: Graefe-Samisch Handbuch der Gesamten Augenheilkunde, vii.

[3] Koester: Virchow's Arch. f. Path. Anat., 1867, xl, 468.

ETIOLOGY

The present knowledge of the etiology of lachrymal gland tumors leaves much to be desired. Acute swelling of one or both glands may be the result of a general infection, such as mumps and erysipelas, or some local infection in some part of the eyeball, or result from traumatism. Chronic swelling of one or both glands may precede or accompany enlargement of the salivary glands and result from tuberculous or syphilitic infection.

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[4] Schenk: Grundriss der normalen Histologie des Menschen.

[5] Weichselbaum: Grundriss der pathologischen Histologie.

Retention cysts, dacryops, arise after inflammatory occlusion of one or more of the excretory ducts, while cystic degeneration is met with in various morbid growths. Dermoid cysts spring from embryonic tissue incorporated in the gland, the same as in other parts of the body, while hydatid cysts are due to echinococci.

DIAGNOSIS AND PROGNOSIS

Save in the purely retention cyst, which is recognized by palpation as a soft fluctuating and perhape lobulated formation, disclosing on eversion of the upper lid a bluish translucent sac, diagnosis is largely conjectural. Resolution under specific treatment is presumptive evidence that we are dealing with gummatous tumor, this especially if both glands were affected. The boundary between simple glandular hypertrophy and adenoma is not a sharp one. Tumors of slow growth, smooth contour and firm consistence are apt to be benign in nature and hence offer a favorable prognosis.

OPERATION

In case of retention cyst, excision of part of the anterior wall through the fornix conjunctiva with gauze drain, should be done rather than an attempt at extirpation of the sac which, because of the thinness of its wall, may be difficult or impossible of accomplishment. For other tumors I perfer extirpation according to the method previously outlined in the relation of my patient, or the outer canthus may be divided and the growth removed through an opening in the fornix conjunctiva.

CONCLUSIONS

All kinds of tumor formations, both of malignant and benign nature, are encountered in the lachrymal gland. In the main the growths are benign in character and of mixed type due to involvement of all the gland tissue, and, according to the preponderance of tissue overgrowth, histologically we may designate them as fibroma, adenofibroma, fibroadenoma or adenoendotheliofibroma. After thorough operative removal we are justified in the majority of cases—this especially where the tumor has been of slow growth—in giving a favorable prognosis recurrences being the exception.

501 Upper First Street.

MEMORANDA

ISOLATED AND COMPLETE PARALYSIS OF THE THIRD NERVE OF TRAUMATIC ORIGIN

REPORT OF A CASE *

LEE MASTEN FRANCIS, PH.B., M.D.
BUFFALO, N. Y.

Alexander Duane[1] makes the following classification of innervational paralysis of the ocular muscles:

1. Extracranial (orbital or peripheral).
2. Intracranial, comprising:
   - A. Basal and foraminal paralysis.
   - B. Nuclear and fascicular paralysis.
   - C. Supranuclear and cortical paralysis.

We are concerned with Group A, namely, basal and foraminal paralysis.

By basal paralysis we understand one due to lesion of the nerve trunk after its emergence from the brain and before its exit from the skull; by foraminal paralysis, one due to lesion of the nerve as it passes through the sphenoidal fissure.

It is to foraminal paralysis that I wish to direct attenby means of the following case:

AUTHOR'S CASE

For the patient who furnishes the text of this paper I am indebted to Dr. J. W. Putnam of Buffalo, who referred her to me on Oct. 14, 1906.. The record is as follows:

History.—Miss E. W. was 17 years old. Her father had died sixteen years before from some malignant disease of the throat. Her mother was living and well. Two older brothers and one older sister were all in splendid health. There was no neurotic strain in the family so far as known. The patient had had no diseases of childhood except measles when quite young; her menstrual history was normal. She had always been in good health with the exception of a suppurating appendix which necessitated surgical interference eighteen months before the present illness. Her convalescence was rapid and complete. Rheumatic tendencies and attacks, sore throat and recent exposures to cold were entirely absent. There was no history of nervous, gastric or intestinal disturbance; no migraine, no headaches, except after prolonged near use of the eyes. Vision had always been acute.

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[1] Chapter 5, Posey and Spiller's The Eye and the Nervous System, Philadelphia, 1906.

Present Illness.—Three evenings before the examinatioon, while preparing to attend a reception at a neighboring house, the patient "bumped" her head against a half-open door as she passed from one rooom to another. The point of injury was at the outer angle of the left supraorbital margin. The force of the blow was not enough to cause more than a passing sting, nor enough to abrade the skin. Disfigurement from possible swelling being feared, a few cold cloths were applied. The patient stated positively that she was neither nauseated, dizzy nor made uncomfortable in any other way, and was equally sure there was no nasal hemorrhage. In fact, the injury was regarded as negligible. Within a relatively short time the patient left the house and attended the reception. A few moments after her arrival a companion asked her why she did not open her left eye. This, the patient thought was about forty-five minutes, and certainly not more than an hour after her injury. The question of her friend was the first intimation of trouble, and it was not until then that the patient discovered her absolute inability to raise the left upper lid. More cold applications were used. The next morning, the ptosis still persisting, the family physician was called, who immediately sent the patient to Dr. Putnam. Putnam's diagnosis was third nerve paralysis; he sent the patient to me for further study, with the assurance that hysteria could be eliminated as a possible etiologic factor, he having gone over the case with this in mind, and also that there were no symptoms of fracture of the skull.

First Examination.—The patient was a well-nourished girl, brunette, and in apparent good health; not of the nervous type.

Right Eye: Vision 6/7.5. Upper lid moved freely. Excursions of the globe perfect in all directions. Pupil normal in size (3 millimeters), reacting readily to light and distance. Accommodation normal. The patient read Jaeger No. 1 readily. Field limits (taken roughly), normal. Fundus negative.

Left Eye: No sign of injury about the orbit. No swelling of the lids or evidence of orbital inflammation or infiltration. Complete ptosis of the upper lid. Globe turned outward and slightly downward. Pupil dilated (6.5 millimeters) and immotile.

Accommodation: Paralyzed. Candle test showed diplopia in all fields except downward and outward to the left. Neither the external rectus nor the superior oblique were involved. Vision 6/45 (plus lenses' aid). Field limits (taken roughly) normal. No scotomata. Fundus negative.

Examination of the nose and accessory sinuses, negative. Hearing normal. Urine examination negative.

A diagnosis of isolated and complete paralysis of the left third nerve was made. The patient's hygiene was regulated, and the faradic current applied three minutes daily.

Course of Disease.—Oct. 20, 1906: Ptosis disappearing. Pupil reacted feebly and was less dilated. Some return of motion. Accommodation returning.

Nov. 5, 1906: Ptosis hardly noticeable. Left pupil less dilated (4.5 millimeters). Divergence much less. Accommodation much improved. At this time the visual fields were taken on the perimeter and both were found to be normal for white and colors. (There was no color inversion.)

Nov. 6, 1906: Refraction under homatropin:

O. D. V. = 6/7.5 + 1.25 sp. c. + 0.25 Cy. axis 90 V. = 6/5.

O. S. V. = 6/15 + 2.75 sp. + 0.25 Cy. axis 90 V. = 6/6.

Retinoscope checks.

Dec. 15, 1906: Ptosis had disappeared. Pupil normal size, responding readily to all reflexes. Accommodation normal. (The patient read Jaeger No. 1 at fifteen inches without effort.) No diplopia except when patient was very tired, at which time the left lid also drooped. Maddox rod showed 15 degrees exophoria.

April 24, 1908: The patient returned complaining of headache, especially on the left side. These headaches were directly associated with near use of eyes. The patient had become a student of stenography.

Second Examination.—No ptosis,, pupils normal in size, equal, responding readily to all reflexes. Accommodation normal. Maddox rod showed 10 degrees exophoria. Refraction under hematropin:

O. D. + 1.25 sp. + 0.25 cy. axis 90 V. = 6/5.

O. S. + 2.00 sp. + 1.00 cy. axis 45 V. = 6/6.

Checked by retinoscope and ophthalmometer.

April 25, 1908. Headaches had disappeared under lenses ordered.

March 14, 1909: The patient returned at my request for examination. She said she had been very well; had had no diplopia, but complained of left headaches which she thought came from her eyes.

Third Examination.—No ptosis. Pupils equal, normal in size and responded readily to all reflexes. Accommodation normal. Maddox rod showed 8 degrees exophoria in distance and

11 near. Candle tests revealed no paralysis or paresis of ocular muscles.

Refraction under homatropin:

O. D. + 1.25 sp. + 0.25 cy. axis 90 V. = 6/5.
O. S. + 2.25 sp. + 0.25 cy. axis 45 V. = 6/6.

Checked by retinoscope and ophthalmometer.

Fundus negative.

March 16, 1909: Fields taken on perimeter were normal for white and colors.

March 18, 1909: Patient telephoned that headaches had ceased under new correction.

At no time during the course of these diseases were there violent headaches or other symptoms of a deep-seated lesion.

In passing, it is interesting to note the refraction change which took place in the left eye. The first cylinder was at axis 90 and was + 0.25. Six months later 1 diopter of astigmatism axis 45 degrees had appeared which in turn diminished to a + 0.25 axis 45 one year later.

As to the location of the lesion in this case, it seems to me that one may eliminate with great certainty the question of fracture of the base. The patient was studied carefully with that hypothesis in mind, without the discovery of a single sign or symptom leading to that diagnosis, unless it was, indeed, the palsy of the third nerve. The patient was also carefully looked over with the view of eliminating the hysterical element. Both Dr. Putnam and myself kept this possibility in mind, but in the face of most thoughtful consideration such a diagnosis was discarded. With equal assurance one may eliminate trouble in the orbit, both on account of the absence of physical signs of damage there and because the branching of the nerve in the orbit renders traumatism producing complete paralysis practically impossible. Therefore the injury must have been either intracranial or at the point where the nerve passes through the sphenoidal fissure. In the absence of any symptoms pointing to intracranial trouble, we must select the sphenoidal fissure as the point of injury. The nature of the process might be either a bruising of the nerve trunk or a subperiosteal hemorrhage of sufficient size to encroach on the nerve but not large enough to injure the other structures, gaining entrance into the orbit through this opening. It must be assumed that the branching at the nerve took place at or near the fissure. In point of the former hypothesis I quote from M.

Allen Starr's[2] text-book the following: "Severe falls and blows on the head have been known to cause bruising of the nerve against the sharp edge of the sphenoidal fissure." Fracture of the wing of the sphenoid at the fissure must also be considered. From the viewpoint of the symptomatology it seems that the subperiosteal hemorrhage hypothesis is most attractive. The fairly sudden onset and gradual clearing up of the symptoms might easily correspond to the absorption of a blood clot.

CASES IN LITERATURE

A systematic and thorough canvass of the literature since 1870, extending through the *Index Medicus,* Index-Catalogue of the Surgeon-General's Library at Washington, current ophthalmologic journals, many text-books, both ophthalmologic and neurologic, and a careful search of Nagel's *Jahresbericht der Ophthalmologie* for the past thirty-nine years, convince one that isolated and complete traumatic paralysis of the third nerve of neither intracranial or orbital origin is rare. I have been able to find the following nine cases, which I have briefly abstracted:

Case 1.—M. Fleury de Clermont[3] in 1862 reports a case of paralysis of the third nerve following a fall on the head.

A farmer, 24 years old, fell from a wagon, striking his left temple on the ground. Complete and isolated paralysis of the left third nerve followed. Recovery was rapid under no other treatment than blistering. There was no evidence of fracture of the skull. (This case is quoted from Chevallereau[4].)

The following six cases are reported from Girardot:[5]

Case 2.—Reported by Desgouttes and Mueller.[6] Antoine C., aged 41, fell, striking on the right parietal eminence. Total isolated paralysis of both third nerves resulted. There was no evidence of fracture of the skull. Recovery took place in one month.

Case 3.—Reported by Desgouttes and Mueller. Julian G., 18 years old, fell on the head, striking on the right occipital region. Loss of consciousness and complete isolated paralysis of both third nerves followed. There were no signs of fractured skull. Recovery took place in three weeks.

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[2] Organic Nervous Diseases, Philadelphia, 1907, p. 647.

[3] Fleury de Clermont: Bull. Soc. de chir., séance du 31 Dec., 1862.

[4] Chevallereau, Armand: Recherches sur les paralysies oculaires consécutives a des traumatismes cérébraux, Paris, 1879.

[5] Girardot, Charles: Paralysies traumatiques isolées et complètes du nerf moteur oculaire commun, Lyons, 1903.

[6] Revue gén. d'ophth., April, 1903, p. 152.

Case 4.—Reported by Durand and G. Gayet.[7] Augustine P., 8½ years old, fell on the head. Loss of consciousness followed and complete bilateral and isolated paralysis of third nerve appearing in one and a half hours. There was no fracture of the skull. Recovery took place in sixteen days.

Case 5.—Reported by M. Ferron.[8] M. G., age not given veterinary surgeon, fell from a horse. Bilateral paralysis of third nerve followed, but disappeared rapidly except slight diplopia and lowered accommodation persisted. There were no evidences of fracture of skull.

Case 6.—Reported by Rothe.[9] Miss D., aged 17, fell from a horse. Loss of consciousness and complete and isolated paralysis of third nerve ensued. There was no fracture of the skull. Complete recovery took place after four months.

Case 7.—Reported by M. Ferron[8]. William F., aged 43, fell on his head. Loss of consciousness followed. There was no evidence of fracture of skull. Complete and isolated third nerve paralysis resulted. Recovery was complete after three months.

Case 8.—J. E. Weeks[10] reports the instance of a young man who was struck one-half inch below the inner canthus on the right eye by a fencing foil, which had pierced his mask. The wound was slight. There was no loss of consciousness. On the following day there was complete paralysis of all structures supplied by the right third nerve. In his remarks concerning the pathology of this case Dr. Weeks says that he thinks it very probable that there was a subperiosteal hemorrhage at the place where the nerve passes through the sphenoidal fissure. Whether there was a fracture of the wing of the sphenoid at this point or whether a rupture of a subperiosteal vessel he does not attempt to decide. Recovery took place in about fifty days.

Case 9.—M. Allen Starr[2] speaks of a patient in his own observation who had a sudden third nerve paralysis following a fall on the head while alighting from a carriage. After six months' duration recovery was complete.

In all the cases quoted above, with the exception of Case 9, in which no detailed history is given, the individuals were in good health with no luetic histories.

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[7] Arch. prov. de chir., March 3, 1901, p. 325.

[8] Thesis, Lyons, 1901.

[9] Memorabilien, 1876, xxi, 411.

[10] A Case of Injury to the Motor Oculi Nerve Followed by Paralysis, New York Ophth. Rec., 1897, vi, 174.

DOUBTFUL BUT POSSIBLE CASES IN LITERATURE

In addition to the nine cases quoted I have found in the literature the following five cases, which are possibly to be grouped as true cases, but which present features making one suspect etiologic factors other than foraminal paralysis. That of Goldschmidt[11] was placed by Girardot on his list, but seems to me to be doubtful on account of the difficulty of eliminating basal fracture with certainty.

CASE 1.—Reported by Goldschmidt.[11] A young man, aged 21, suffered a gunshot wound in the mouth. Several hours later complete and total paralysis of the third nerve presented itself. Goldschmidt does not speak of the result or say whether there were symptoms of basal fracture. (Classed with doubtful cases on this account.)

CASE 2.—Reported by F. Denti.[12] A man, 38 years old, while intoxicated, fell from steps and sustained a total paralysis of the right ocular motor. (There was no report as to whether there was involvement of other nerves or as to whether there was fracture of the skull.)

CASE 3.—Reported by Gaupillat and Regnault.[13] A young soldier, while fencing, was struck in the inner angle of the eye by a bayonet, making a very slight tearing of the conjunctiva. The patient was much depressed, fearing loss of sight. The eye was immovable; the pupil dilated; accommodation absent; light perception only. All unfavorable symptoms passed away and the question as to hysteria as the factor arose. (The loss of vision makes a diagnosis of hysteria possible, especially when taken with the patient's mental depression.)

CASE 4.—Reported by Prof. J. Komoto.[14] A boy 4 years old was injured twenty-five days previous to having been seen. He was struck on the upper left side of the forehead by a falling wall-clock. There was no loss of consciousness or vomiting. There was a left ptosis. The eye turned out and down. The pupils were normal. Recovery, except slight ptosis, took place. On account of the non-involvement of the pupil the diagnosis of nuclear paralysis is probable. It is possible that had the case been seen immediately after the injury, paralysis of the contractor fibers of the iris would have been noted.

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[11] Wien. med. Wchnschr., 1893, xliii, No. 7.

[12] Paralisi completa, periferica, traumatica del ter 20 pago del nervi cranici di destra, Ann. di Ottal., xii, 562.

[13] Clin. Ophth., Feb. 25, 1908.

[14] Nippon Gankwa Gakukwai Zasshi, viii, No. 1.

Case 5.—Devoe[15] reports a case of double third nerve paralysis following an accident. Devoe believed that the cause was hysterical. Recovery. (The details of this case are so meager that it is impossible to class it definitely. Fracture of the skull is not eliminated or paralysis of other ocular nerves.)

In conclusion I can do no better than translate verbatim the conclusions of Dr. Girardot's admirable thesis on this subject:

1. Traumatic paralysis of the motor oculi are rare, especially when one takes into consideration the frequency of palsies of the ocular muscles.
2. The anatomic relations of the motor oculi with the base of the brain are such as to make laceration or compression by bone lesions easy. Indeed, it is not uncommon to see paralysis of the motor oculi associated with those of the facial, or patheticus or abducent and following fractures of the skull, but the isolated paralysis of the third nerve are independent of fractures. They are, probably, due to a hematoma located about the nerve or in its sheath.
3. From the standpoint of the symptomatology there is nothing of special interest, except that they are isolated and complete.
4. Admitting compression by hematoma it does not seem that surgical interference is justifiable, for though the process be a slow one, recovery occurs spontaneously in two months.

482 Delaware Avenue.

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[15] Semaine méd., 1890.

MEMORANDA

MEMORANDA

GALVANOCAUTERY PUNCTURE IN ECTROPION AND ENTROPION *

S. LEWIS ZIEGLER, A.M., M.D.
Attending Surgeon, Wills Eye Hospital, Ophthalmic Surgeon, St. Joseph's Hospital
PHILADELPHIA

Ectropion and entropion, of mild degree, whether from interstitial shrinkage or from moderate cicatricial contracture, are so inefficiently remedied by our present operative measures that I have thought it desirable to present to the profession a simple procedure I have used for many years and from which I have never yet failed to secure the desired results.

There are many conditions which we can relieve by this procedure:

A. Spastic entropion or ectropion.
B. Senile or relaxed ectropion or entropion.
C. Paralytic ectropion.
D. Contraction from lachrymal irritation.
E. Moderate cicatricial contraction.
F. Small cicatricial distortions or dimplings.
G. Cases of distichiasis.

Many varieties of suture operations have been employed to relieve these conditions, but, while successful in some instances, they are quite inefficient in the larger proportion of cases.

The plastic operations often leave a scar. which is more disfiguring than the original lesion, and is, therefore, distasteful alike to both patient and surgeon.

The use of flexible collodion has proved of some value in spastic entropion following operations requiring the use of a compress bandage. A few galvanocautery punctures, however, will prove far more efficient in relieving this class of cases.

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INSTRUMENTS

The method I desire to propose requires but two instruments, a short galvanocautery point (Fig. 1), and a lid clamp (Fig. 2). The galvanocautery point should be short, rather thick and sharpened at the point so that it will puncture quickly. This avoids scarring. The lid clamp is a modification of my chalazion clamp, having a straight edge, which is a perfect guide for the row of punctures. It is made with a sliding catch, instead of the Desmarres screw, which facilitates placing it in position. The clamp makes a bloodless operation, and, to a certain degree, benumbs the tissues and so lessens the pain. If no clamp is available, the ordinary Snellen lid spatula may be slipped under the lid and the margin of the lid held flat by the fixation forceps or by a double lid hook, in cases of entropion. The same method may be reversed in ectropion, the lid

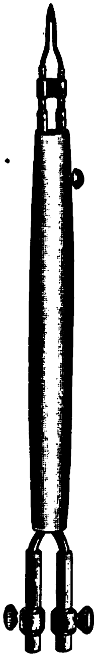

Fig. 1.—Galvanocautery handle and tip for galvanocautery puncture operation in ectropion and entropion.

spatula being placed outside on the skin surface, and the margin drawn away from the eye by the forceps or lid hook, which is then turned back flat over the lid spatula.

ANESTHESIA

I usually employ nothing but 4 per cent. cocain on the conjunctival surface, the pressure of the clamp causing some anesthesia. The patient may wince a little because of the glow of the cautery, the slight smoke and the momentary noise of sizzling or frying flesh, but ordinarily very little complaint is made.

In nervous, sensitive patients deeper anesthesia is required. Hypodermic injections of cocain under the skin, or conjunctiva will often increase the local anesthesia. In entropion the skin of the lid may be slighty frozen by a spray of ethyl chlorid. In the majority of cases, however, it will be necessary to employ nitrous oxid. bromid of ethyl, chloroform or ether. If ether is used it must be removed before the approach of the hot cautery point or an explosion will naturally occur.

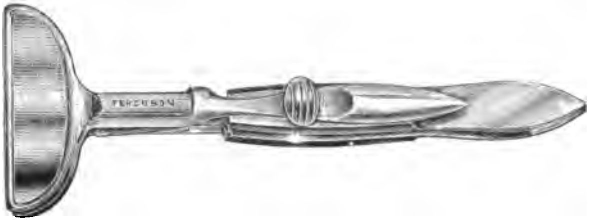

Fig. 2.—Lid clamp for use in galvanocautery puncture of the eyelids. Can be used for both eyelids, either on conjunctival or skin surface.

DESCRIPTION OF OPERATION

The lid clamp is adjusted with its straight bar 6 mm. from the lid margin (Figs. 3 and 4). The galvanocautery point is applied to the surface with considerable pressure, the button on the handle is pressed down to turn on the current, while the point is quickly pushed through the cartilage and as quickly withdrawn. The punctures are made 4 mm. from the lid margin, and separated from each other by an equal interval of 4 mm. These should be made on the side on which we wish the

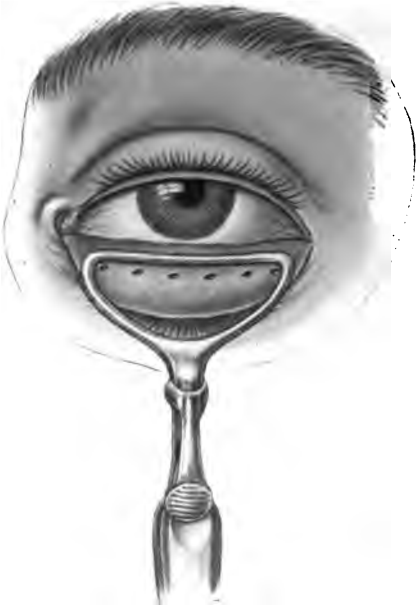

Fig. 3.—Lid clamp adjusted to case of ectropion, showing row of galvanocautery punctures.

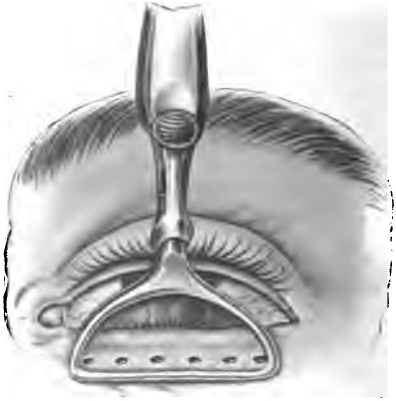

Fig. 4.—Lid clamp adjusted to case of entropion, showing row of galvanocautery punctures.

contraction to take place, viz., the conjunctival surface in ectropion, and the skin surface in entropion. If necessary, we can repeat the procedure in a few weeks. From one to three sittings will accomplish as much as a plastic operation would do.

This procedure possesses the double advantage of causing vertical contraction of the relaxed lid, and at the same time producing shrinkage and shortening of the redundant length of the lid, thus restoring the lid to its normal position.

POSTOPERATIVE REACTION

I have seldom seen any reaction. If, however, there should be cellulitis and puffing of the lid, the application of continuous ice pads for a day or two will control this condition. I have seen one case (see Case 10) infected by the discharge of a purulent dacryocystitis. I controlled this by wrapping a small wisp of cotton on a wire applicator, dipping it into a 1 per cent. formalin solution, and twirling it around slowly in each puncture. This application was made for three successive days, in addition to the ice padding, when all signs of sloughing disappeared and healing occurred without a scar. Every case, of course, exhibits the charred eschar of the cautery but this causes no disturbance and usually clears off in about a week.

REPETITION OF THE PUNCTURE

It is generally necessary to repeat the operation two of three times at intervals of from two to four weeks, depending entirely on the amount of incurvation or turning out to be overcome. As the procedure is under perfect control, the judgment of the operator can easily determine the number of punctures necessary to attain the desired result. In cases requiring repetition it will be better to make the punctures alternate, locating the second series between the first series.

REPORT OF CASES

I desire to relate briefly a sufficient number of cases properly to illustrate the lesions under consideration and to demonstrate the value of the galvanocautery procedure. The majority of these cases were treated at the Wills Eye Hospital, while a few were seen at the St. Joseph's Hospital or in private. I regret that more photographs of the cases were not taken in order to show more especially the results of the operation.

Case 1. C. S. P., aged 75, suffered from senile or relaxed ectropion of lower lid, O. S., with lachrymal obstruction.

Oct. 9, 1898: Rapid dilatation, O. S.

Dec. 11, 1898: Galvanocautery puncture of conjunctival surface, lower lid, O. S.

Jan. 8, 1899: Galvanocautery puncture, O. S., repeated.

Feb. 1, 1899: Final galvanocautery puncture, O. S., which was followed by prompt and complete recovery.

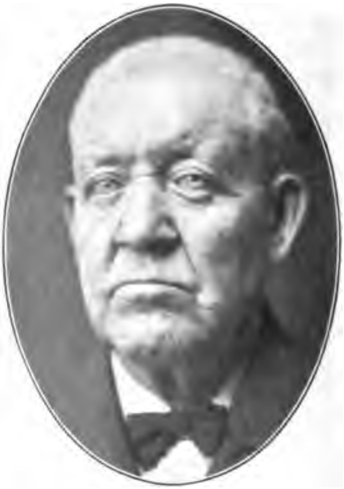

Fig. 5 (Case 3).—Spastic entropion following cataract extraction.

Case 2. W. T., aged 29, ectropion of lower lid from lachrymal irritation; obstruction of tear duct.

Nov. 1, 1905: Galvanocautery puncture of lower lid, O. D., and rapid dilatation of tear duct.

Dec. 9, 1905: Galvanocautery puncture, O. D., repeated.

Recovery was prompt and complete.

Case 3. J. B., aged 66, developed senile or relaxed ectropion of the lower lid, O. S., after cataract operation. This was spastic in character.

Dec. 9, 1905: Galvanocautery puncture, O. S., followed by rapid recovery which has been permanent. The original condition is well shown in the cut, Figure 5.

Case 4: T. R., aged 42, ectropion of lower lid with lachrymal obstruction, blepharitis and epiphora, O. S.

Jan. 3, 1906: Galvanocautery puncture, O. S., followed by Stilling's operation and insertion of lead style. The recovery was prompt and complete.

Case 5. F. T., aged 46, cicatricial entropion of lower lid, O. S., following burn by acid, which also occluded the tear duct.

Jan. 11, 1906: Stilling's operation with lead style O. S.

Jan. 17, 1906: Galvanocautery, puncture, O. S., which drew the lid out to its normal position, the convalescence being very rapid.

Case 6. W. H. B., aged 48, ectropion of lower lid, O. S., from lachrymal irritation, with occlusion of tear duct.

May 14, 1906: Galvanocautery puncture, O. S.

May 29, 1906: Puncture repeated.

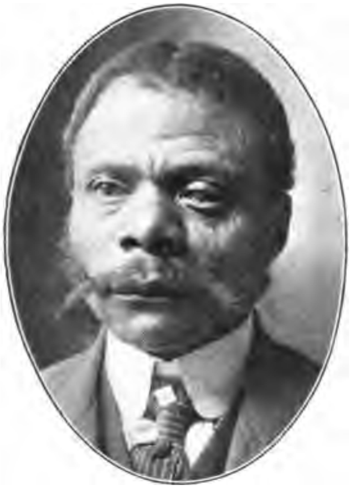

Fig. 6 (Case 6).—Ectropion from lacrimal irritation.

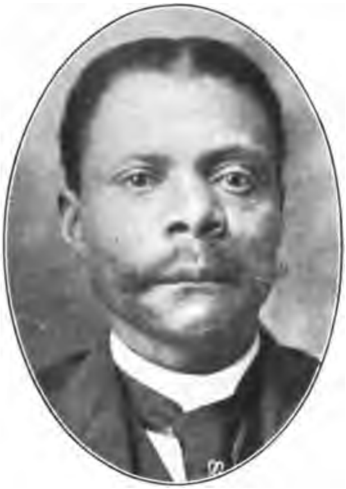

Fig. 7 (Case 6).—The same patient two years after operation.

June 25, 1906: Rapid dilatation of the tear duct, O. S. Figure 6 shows the condition before operation, May, 1906, while Figure 7 represents the condition two years later, showing that the result has been complete and permanent.

Case 7. C. H. A., aged 35, O. D. was injured by molten metal and enucleated. The patient had relaxed ectropion of lower lid, O. D., with shallow socket.

May 27, 1907: Galvanocautery puncture, O. D., conjunctival surface.

Nov. 4, 1907: Galvanocautery puncture repeated, which brought lid into good position for wearing an artificial eye.

Case 8. A. L., aged 33, entropion with trichiasis and lachrymal obstruction.

Nov. 1, 1907: Rapid dilatation, O^2.

Nov. 11, 1907: External canthotomy, O^2.

Nov. 22, 1907: Galvanocautery puncture of skin surface, which relieved the entropion and trichiasis.

Case 9. D. S., aged 75, entropion with trichiasis of both lids, O^2, the sequela of trachoma; lachrymal discharge with pannus, xerosis of conjunctiva.

Nov. 11, 1907: Galvanocautery puncture of skin surface, O^2.

Dec. 18, 1907: Galvanocautery puncture, O^2, which so completely corrected the entropion and trichiasis that I was able to do a combined cataract extraction in O. S. two months later.

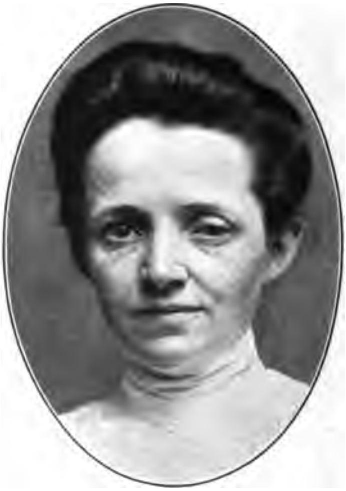

Fig. 8 (Case 13).—Entropion and trichiasis before operation.

Case 10. T. T., aged 55, had both eyes burned by an explosion thirty years before the time of operation; had entropion, trichiasis, dacryocystitis and pannus in both eyes; also had traumatic pterygium, O^2, with cicatricial bands in O. D., which held the eye in a divergent position.

April 1, 1908: Pterygium excised; Stilling's operation with lead style in both eyes.

April 6, 1908: Galvanocautery of skin surface for entropion of upper lid, O^2.

April 22, 1908: Division of cicatricial bands of upper lid by galvanocautery incision, O. D., which freed the eye from its divergent strabismus.

April 24, 1908: Galvanocautery puncture repeated in both eyes.

May 8, 1908: Galvanocautery puncture repeated, followed by infection from dacryocystitis, which was controlled by applications of 1 per cent. formalin solution, and ice padding. This is the only case of infection I have noted.

The ultimate result of these various procedures was excellent, and the patient was discharged greatly improved in appearance, with better vision and greater comfort than he had enjoyed for many years.

Case 11. S. M., aged 67, has marked senile ectropion of lower lid with blepharospasm, O. D., dating back one year.

April 24, 1908: Galvanocautery puncture, O. D., conjunctival surface.

May 1, 1908: Galvanocautery puncture, O. D., repeated.

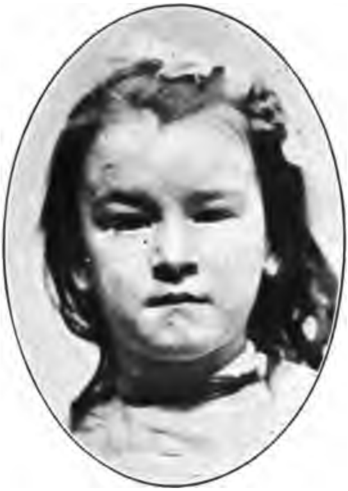

Fig. 9 (Case 14).—Congenital entropion before operation.

May 13, 1908: Galvanocautery puncture, O. D., the final result being a restoration of the lid to its normal position with relief of the blepharospasm.

Case 12. W. W. G., aged 49, spastic entropion of lower lid, O. S., with lachrymal obstruction and ulcerative keratitis.

May 28, 1908: Galvanocautery puncture of lower lid, O. S., with rapid dilatation of the tear duct. The convalescence was prompt and complete.

Case 13. Miss E. G., aged 34, suffering from entropion and trichiasis of upper lid, O. S., for many years.

June 10, 1908: Galvanocautery puncture for entropion of upper lid, O. S.

June 24, 1908: Operation repeated.

Figure 8 shows the condition prior to operation. The result was excellent but, unfortunately, the patient was discharged from the hospital without securing a photograph showing the final condition.

CASE 14. Miss R. G., aged 9, congenital entropion of both lids of each eye, with complete inversion of eyelashes; lachrymal obstruction.

Feb. 17, 1908: Rapid dilatation of both tear ducts.

May 25, 1908: Galvanocautery puncture of both lids, O^2.

June 29, 1908: Operation repeated on both eyes. Marked improvement.

July 8, 1908: Final puncture, which was followed by complete relief. Examination of the patient on March 8, 1909, showed that the result was permanent.

Figure 9 represents the condition before operation.

1625 Walnut Street.

MEMORANDA

MEMORANDA

THE OPERATIVE TREATMENT OF GLAUCOMA BY CYCLODIALYSIS *

ARNOLD KNAPP, M.D.
NEW YORK

The operation of cyclodialysis suggested by Heine in 1905, consists in making an artificial communication between the anterior chamber and the suprachorioidal space, allowing a new channel for the escape of the acqueous humor and thus reducing intraocular tension. The operation is performed under local anesthesia by making an incision in the sclera 6 to 8 mm. posterior to the sclerocorneal junction after reflecting a conjunctival flap and with a bent spatula, separating the ciliary body from the overlying sclera and breaking through the pectinate ligament. A quadrant of the iris periphery is then detached. I have performed this operation in eighteen cases specially selected to test its merits. These cases have now been observed for a sufficiently long time to permit an opinion on the value of this operation.

The cases can be arranged in the following groups:

A. CHRONIC GLAUCOMA

These are cases in which an iridectomy did not succeed in permanently reducing the tension, or a contraindication to an iridectomy was present.

CASE 1.—R. B. M. Age 55. L. Vision=20/20—. T. (Tension)+1. Fn. (Field normal) Nov. 14, 1905; Iridectomy. T. again increased after six weeks. June 2, 1906: Cyclodialysis. At operation some difficulty in breaking through pectinate ligament. Aug. 1, 1906: Tn. V.=20/30. Dec. 1, 1906: Some opacities in posterior cortex of lens. Tn. (Tension normal) March 22, 1909: T. has remained normal without any miotics; cataract nearly mature.

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Case 2.—E. M. R. Age 60. Aug. 1, 1905: Iridectomy. Notwithstanding drops, eye showed tendency to be hard. V. = 20/30. F. contracted nasally and below. May 21, 1906: Cyclodialysis. During operation anterior chamber filled with blood. This was evacuated by paracentesis two days later. V.=20/40. On the last visit, May 6, 1908, V.=20/30; F. no change. Tn. Has occasionally used miotics.

Cases 3 and 4.—S. G. Age 45. R. absolute glaucoma. L. 20/100. T.+. F. contracted nasally to 10 degrees. March 20, 1906: R. cyclodialysis. April 1, 1906: No reaction. No chorioidal detachment appreciable. Tn. April 4, 1906: L. cyclodialysis. April 20, 1906: R. T.+; L. Tn. March 10, 1907: R. T.+. L. 20/70. F. no change. T+½. To use drops. April 3, 1908: L. tension kept slightly elevated, hence cyclodialysis repeated in left eye. F. nasally contracted to 5 degrees. Incision in sclera external to old one. On introducing spatula instrument passed into anterior chamber without encountering any resistance. June 17, 1908: L. Tn. 20/70. R. T.+. Dec. 30, 1908: L. Tn. Feb. 24, 1909: L. Tn. R. T.+.

Case 5.—M. W. Age 56. Marked arteriosclerosis. R. widely dilated pupil. T.+. Od. cup with atrophy. V.=20/100. F. nasal contraction to 5 degrees. May 2, 1907: Cyclodialysis. Uneventful. June 17, 1908: R. 20/200. Tn. No miotics. March 2, 1909: Tn. V.=20/200. No drops.

Case 6.—P. P. Age 61. R. iridectomy, 1905. May 1, 1907: R. V.=20/200. F. 15° N. (Nasal) 30° temp. T.+ Cyclodialysis. Uneventful, June 10, 1908: R. 20/200—; T.+?. F. Slightly more contracted. March 8, 1909: T.+.

Case 7.—A. G. Age 42. R. 20/200. F. 80 degrees temp; 35 degrees sup. 35 degrees inf. 30 degrees nasal. T.+. Aug. 27, 1908: Cyclodialysis. During operation anterior chamber filled with blood. That evening attack acute glaucoma, relieved by hot applications, eserin (physostigmin) and dionin. Nov. 28, 1908: T.+. Arc of iris detached showed atrophy of stroma. Feb. 24, 1909: T.+.

Cases 8 and 9.—J. S. (R. od.) cup with atrophy. July 31, 1908: R. cyclodialysis. Aug. 7, 1908: L. cyclodialysis; both uneventful. Aug. 20, 1908: T.— in both. R. V.=m. h. (movements of hands) L. V.=20/70. F. 25 degrees nasal. 30 degrees sup. 10 degrees inf. 45 degrees, temp. September 9, 1908: Tn. each. Oct. 12, 1908, last visit, Tn. each.

Case 10.—F. W. Age 40. L. od. cup with atrophy. V.= hand movements in temporal field. T.+ Sept. 29, 1908: Cyclodialysis. Some hemorrhage and posterior adhesions. Oct. 13, 1908: T.—. Small hemorrhage on disc. November 27, Tn. F. had enlarged. March 29, 1909: L. opac. vitr. T.—. V. no change. F. enlarged.

Case 11.—R. F. Age 50. L. iridectomy two years ago. Deep cup with atrophy. V.=20/100. F. contracted to small temporal area. T.+. June 27, 1908: Cyclodialysis. Uneventful. Feb. 20, 1909: V. 20/70. Tn. Field enlarged.

Case 12.—T. C. A. Age 60. Feb. 27, 1907: L. V.=10/200. F. contracted to temporal half. T.+. Nov. 27, 1907: Cyclodialysis. Uneventful. T. reduced. Optical iridectomy later for old opacity of cornea. April 4, 1908, last visit, 10/200. Tn.

Case 13.—A. S. Age 62. Chronic glaucoma with atrophy. L. 20/70—. T.+. Dec. 2, 1908: Cyclodialysis. Very difficult to enter anterior chamber. Jan. 26, 1909: Last visit. L. 20/100. Field enlarged out and down. Tn. (no miotics).

Cases 14 and 15.—M. L. Age 26. V. always poor, especially for last six months. R. 20/200. T.+. F. irregular contraction. L. m. h. T.+. F. contracted to temporal and superior quadrant. Very deep cup. Pronounced atrophy. Dec. 18, 1907: R. cyclodialysis. T. reduced and remained minus. Dec. 26, 1907: L. cyclodialysis. After 1 week, T. rose again. Jan. 6, 1908: Repeated operation. T.— since. Jan. 20, 1908: R. 20/200. L. 20/200. Fields enlarged. Aug. 28, 1908: R. T.+ again. L. T.+. R. cyclodialysis repeated. Favorable except that it was followed by retinal hemorrhage in macula. Sudden attack acute glaucoma requiring paracentesis. Jan. 7, 1909: R. cyclodialysis repeated. Down and out. T. very soft. Cornea slightly collapsed. V. eccentric 3/200. L. T.— V.= 20/200. F. enlarged.

B. CONGENITAL GLAUCOMA

Case 16.—J. G. Age 1 year. Parents first cousins. At six months diagnosis made. July 1, 1908: Deep cup. Prescribed pilocarpin. Eyes enlarged. Corneæ dull. Cyclodialysis in both. Sclera very thin. No resistance whatever at ligament. Aug. 1, 1908: R. again hard. L. Tn. Mother thought that child could now see. Dec. 30, 1908: Tn. each. Jan. 6, 1909: R. Tn. L. T+. Using pilocarpin 2 per cent.

C. EXTENSIVE CAPSULAR ADHESION TO CORNEA

Case 17.—A. C. Age 30. Dec. 2, 1907: Struck in eye with stick. Transverse wound of cornea. Irideremia. Broad adhesion of lens capsule to corneal scar. T. remained hard notwithstanding all medicinal treatment. Jan. 20, 1908: Cyclodialysis. On passing spatula an obstruction encountered more posterior than usual. A quadrant detached. T. not perceptibly reduced. On following day eye very hard. This continued; tension somewhat reduced by daily pilocarpin sweats. Jan. 26, 1908: T.+. Patient lost sight of.

D. GLAUCOMA SECONDARY TO NEURORETINITIS WITH VASCULAR CHANGES AND RETINAL HEMORRHAGES

CASE 18.—S. Age 46. Acute glaucoma not controlled by miotics, sweats, and mercurial injections. Cyclodialysis. T. reduced for two days, then relapse. Patient lost sight of.

In brief the operation has failed in congenital glaucoma (Case 16), in glaucoma secondary to extensive capsular adhesion to the cornea (Case 17), and in glaucoma secondary to neuroretinitis with endarteritis and hemorrhages (Case 18). It has been partly successful in cases of chronic glaucoma (Cases 1 to 15) as will be seen in the following summary:

CASE 1.—T. remained normal without miotics to present date, a period of two years, nine months.

CASE 2.—T. remained normal with occasional use of miotics to date of last visit, two years.

CASE 3.—T. remained normal for one year then became hard.

CASE 4.—T. remained down for one year and a half, then operation repeated and T. has remained down to present date, eleven months.

CASE 5.—T. has remained down to date, one year, ten months. No miotics.

CASE 6.—T. remained down for one year, has now been somewhat raised for ten months, incompletely controlled by miotics.

CASE 7.—T. reduced for four months. Has now been elevated for three months.

CASES 8 AND 9.—T. down at last visit, two months, without drops.

CASE 10.—T. down now for six months since operation.

CASE 11.—T. down now for nine months.

CASE 12.—T. down during five months' period of observation.

CASE 13.—T. down at last visit, two months.

CASE 14.—T. down for one year, eight months, with enlargement of field; operation repeated then (August, 1908), followed by hemorrhage in macular region, attacks of acute glaucoma; operation repeated in January, 1909. T.— since. V. very much reduced as field as eccentric.

CASE 15.—T. down for one week. Operation repeated. T.— since; period of observation one year, three months. V. and F. improved.

These were all cases selected to test the value of cyclodialysis, in which an iridectomy had been performed with no permanent benefit or in which, on account of the advanced stage of the glaucoma, the atrophy of the optic nerve, the restricted field or the condition of the iris, not much result could be expected from an iridectomy. Of these the tension was reduced in eleven for observation-periods varying from two years and nine months to two months. In the remaining four the tension again became hard.

The operation is technically easy, much easier than a correct iridectomy. The difficulties encountered in breaking through the pectinate ligament differed and were readily recognized. In one in which at the second operation the same quadrant of iris was displaced, no resistance at all was felt as if a firm adhesion had not reformed. In one case this segment of iris subsequently developed distinct atrophy of the iris stroma. In one of the most favorable cases a cataract developed, the opacity beginning in the posterior cortex. It is impossible to say whether the cyclodialysis was responsible for this. The operation did not aggravate the condition except for a macular hemorrhage in one, and attacks of acute glaucoma in two.. No vitreous disturbance or detachment of the chorioid were observed. The operation was repeated with benefit in three cases. In one (Case 14), the eye subsequently exhibited marked hypotony which apparently was not harmful but this again is a point requiring further observation. Why the operation succeeded in some, and in apparently similar cases the tension again rose, can not be explained.

It can be conservatively stated that cyclodialysis is of value in certain limited conditions; it is indicated in the advanced cases of chronic glaucoma, especially those in which iridectomy has not succeeded in reducing the tension. It can not in any way replace the classical operation of iridectomy or its recent modifications in incipient cases of chronic glaucoma.

26 West Fortieth Street.

MEMORANDA

TENOTOMY OR ADVANCEMENT *

LUCIEN HOWE, M.D.

BUFFALO, N. Y.

When Dieffenbach and his contemporaries began to operate for heterotropia they always divided the tendon of the muscle, or even the muscle itself toward which the eye turned. Naturally many of these eyes soon turned quite as far out of place in the opposite direction. Then operations for advancement began to be made, although at first only in a crude form. During the three-quarters of a century since that time ophthalmologists have differed in their opinions as to when to make tenotomy and when advancement. We have the important fact that thousands of eyes have been satisfactorily straightened by tenotomy, and, on the other hand, we know the unsatisfactory results of this operation which come to even the best operators, and the theoretical reason for advancement—that it is always better to strengthen one muscle than to weaken its opponent. An examination of the literature seems to indicate that when some attractive form of tenotomy has been proposed, ophthalmologic opinion swung toward that operation, or when another for advancement offered it swung toward that. Of late the tendency—in America, at least—seems to be in favor of advancement, in spite of the fact that this is by far the more difficult operation for the surgeon to make and the more uncomfortable for the patient to endure.

My object here is to recapitulate briefly the reasons for deciding in a given case on a tenotomy or an advancement. As exact definition is necessary to clear thinking, it should be stated that each of these terms

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is used here in its broadest sense. It is taken for granted that the tenotomy made in a given case is always the form of that operation then indicated, whether that be a partial, a simple, or a complete tenotomy; also that the proper form of advancement is always selected, whether that be advancement of the tendon alone or of the tendon with its capsule, it being far beyond the province of this paper to discuss at all the technic of either operation.

It should be noted also in what follows that the term "strabismus" is avoided, because that is so indefinite as to mean all forms of heterotropia and therefore no one form exactly.

Moreover, even the question of diagnosis here dealt with is presented with some hesitation, as a short chapter on this subject has been given recently in my second volume[1] on the muscles, and there is danger of repetition. But, according to the plan adopted in that book, it was not practicable to arrange the facts in the order which is possible here.

We shall probably all agree at once on the fundamental proposition that the operation selected should be the one which restores most completely the normal condition. Thus if the deviation of an eye is produced by the excessive action of a certain muscle or group of muscles, we are safe in lessening that excessive action by some form of tenotomy. On the other hand, if the deviation is produced by the insufficient action of a certain muscle or group of muscles, advancement is indicated. In other words, the decision in a given case between a tenotomy or an advancement depends on the question whether the deviation is due to an excessive action of the muscle or group of muscles toward which the eye turns, or to the insufficient action of the muscles or group of muscles away from which it turns.

For the sake of clearness we should remember that our cases of heterotropia really divide themselves into three great classes, each class having apparently several divisions of its own—more or less well defined. We have long recognized the more obvious examples of each great class and treated them accordingly. Taking them in the order in which we began to separate them from each other, we recall the cases most easily recognized as those of paralysis or paresis or insufficient action (as we wish to call it) of one muscle or group of muscles. Sometimes the paralysis is apparent at a glance; sometimes it is necessary to have the patient look to the right, left, up or down, to assure ourselves that any paresis exists; while in still other patients that diagnosis can be made only after long and patient search. These we may well call cases of actual insufficient muscular action, and for them we agree that advancement would be indicated.

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[1] Published last year by Putnam, New York.

We should remember, however, that an abnormal condition of the opposite kind may also be observed: a spasm or excessive action (as we wish to call it) of one muscle or group of muscles. If the limits of this paper permitted, the diagnosis, frequency, etc., of these cases would be dwelt on, but I have recently tried to give a summary of our knowledge on that subject. It is only necessary here to observe that the largest and nosis can be made only after long and patient search. Donders long ago called attention. The hypermetropes whose ciliary muscles were constantly in a state of excessive action, and with that came excessive action of the internal recti. These we may call cases of actual excessive muscular action, and for them most of us also agree that tenotomy would be indicated. Without doubt there are other groups of this class with excessive muscle action which still remain to be separated from the rest and carefully studied. This seems probable, at least in view of the thousands of good results blundered into after so-called tenotomies made in the most careless manner possible.

Between these two classes of deviations due only, on the one hand, to excessive, and, on the other, to insufficient action of a muscle or group of muscles, we have a third class, in which these two conditions exist in the same eye. Thus an esotropia very often results from excessive action of the internus, together with insufficient action of the externus. This is the rule in cases of long standing. My main point is simply this: In order to decide in a given case whether to make tenotomy or advancement we must know first of all the nature of the deviation. We must ask, Does that eye turn out of place because of the insufficient action of one group of muscles or because of the excessive action of another group, or because of both causes acting at once? If the last, which cause predominates? Minor questions are also involved in this decision, but they may be neglected for the present.

Moreover, for the present let us limit ourselves to deviations of the visual axes which occur in the horizontal plane; that is, to esotropia and exotropia. They will illustrate the principles involved. To be still more definite, let us imagine for a moment that we are dealing with a case of esotropia in a young person. In deciding a question like this it is necessary to obtain all of the evidence possible from different sources. These sources are:

A. The refraction, with the condition of the ciliary muscles.

B. The extent of the arc of rotation and the behavior of the eye, in rotating, as shown by the perimeter or tropometer or both.

C. The exact character of the arc of rotation as shown by photographic tracings.

D. The lifting power of the adductors and abductors as shown by the ophthalmodynamometer.

Let us consider these in order.

A. THE REFRACTION AND THE CONDITION OF THE CILIARY MUSCLES

The part which hypermetropia plays in esotropia has already been referred to and is too well known to require any elaboration here. It is the deviation inward which Galezowski and others have called functional strabismus. This is a good example of the cases in which the deviation inward is due to excessive action of the adductors. The treatment with convex glasses shows that we recognize this. When that is not sufficient, especially when one eye is amblyopic, then most operators are accustomed to make some form of tenotomy of the internus. Good results have followed in so many thousands of such cases that probably tenotomies will always be made for them, in spite of any theories in favor of advancement.

In a word, the condition of the refraction not only gives us a clue as to the diagnosis but also to the treatment.

B. EXTENT OF ARC OF ROTATION AND BEHAVIOR OF EYE IN ROTATING

Important evidence is furnished by the extent of the arc of rotation in and out, and by the behavior of the eye in moving through that arc. The limits of this paper will not permit a description of the form of perimetèr or tropometer best suited for making these measurements. They have been described at length in the first volume on the muscles in the work already referred to. A more complete description of their varieties, and the advantages of different forms, are to be given in a paper now in press, which will be presented at the meeting of the American Ophthalmological Society to be held in July and published in their transactions. At present it must be taken for granted that these forms of the perimeter and tropometer are well known.

It should be mentioned, however, that while the perimeter is very useful for measuring the extent of the arc of rotation outward, it is of no value in measuring that arc up or down, because of the obstruction to the axis of vision caused by the projection of the nose, brow or cheek. For the measurements of the arc of rotation in these three directions we must depend on some form of the tropometer. The form which I have described, that is, a tangent scale in the tube of the horizontal microscope, seems to serve the purpose as well as the more complicated instruments.

It should also be observed that the extent of the arc of rotation is by no means the only measure of the amount which the eye can turn in a given direction. I have already called attention to the fact, and must repeat again, that quite as much depends on the character of the rotation as on its extent. To say that an eye rotates outward, for example, 50 or 55 degrees, means but little. We must also know whether that rotation is a steady and uninterrupted swing, to a point at which the eye remains fixed as long as the individual desires, or whether, on the other hand, the eye swings out steadily only to 25 or 30 degrees, then begins to halt or vacillate, as it were, and, after reaching even the same piont of rotation, to swing quickly back, in spite of the effort of the individual to hold it in that position. In making any of those measurements with either the perimeter or tropometer we must, therefore, have a suitable instrument, and the observer must also have a familiarity with the instrument before he is able to obtain constant results. Apparently it is almost as difficult to measure accurately the arc of rotation and to estimate properly the character of the rotation of an eye as it is to learn the manipulation of an ophthalmoscope.

C. EXACT CHARACTER OF ARC OF ROTATION

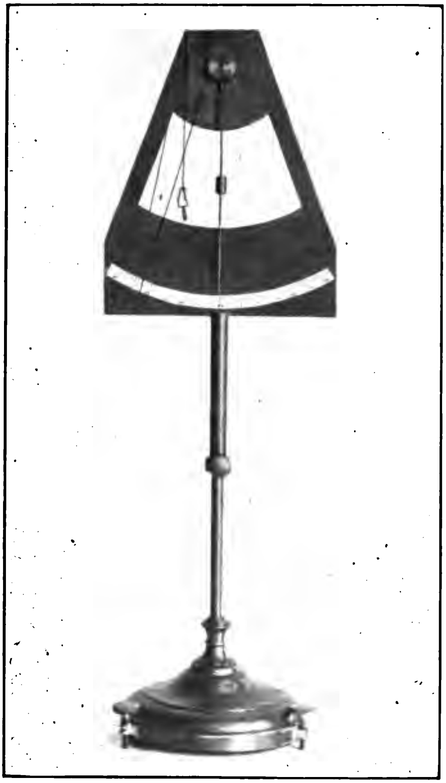
More exact testimony as to the extent and character of the arc of rotation and its rapidity is shown by photographic tracings of the reflex from the moving cornea, or what has been described as photograms. These are considered next in order only because the testimony furnished by them is similar to that given by the perimeter and the tropometer. They all deal with the arc of rotation. As the photographic plate shows to the astronomer the existence of stars which no eye has ever seen with the largest telescope, so does the plate also record the extent of the arc of rotation of an eye and the variations in its movements more exactly than can be seen by the eye of any observer when using a perimeter or tropometer.

The limits of this paper again make it impossible to elaborate this point, but I have already published the method for making these photograms both by daylight and, still better, by artificial light. They are referred to here, as already stated, because this is a more exact method of measuring the arc of rotation.

It should be understood, of course, that it is not a method of any value for every-day office work. The clinician ordinarily has too much to do otherwise to take time for such details. It should be observed, however, that the fact that he is busy has nothing to do with the question at issue. We are now considering careful, honest work. While it is true that such measurements can never become general, it is true also that they do assist somewhat in deciding in a given case whether to make a tenotomy or an advancement. For this reason, if for no other, it is probable that this method of diagnosis in some form will ultimately come into use, at least in large institutions, just as we resort there to the use of the x-ray or the giant magnet.

D. THE LIFTING POWER OF THE ADDUCTORS AND ABDUCTORS

Finally some light is thrown on the character of a given deviation by the lifting power of the adductors and abductors. Here again a long digression would be necessary to describe the method of making these measurements. The earlier arrangements for that purpose were outlined in the first volume of the work on the muscles already referred to.



Ophthalmic dynamometer for measuring the lifting power of the horizontal recti

Since that was published a decided improvement has been made in the technic. That was described in a paper I presented at the International Congress in Naples in April. It may be stated in explanation that after cocain has been applied, a speculum is introduced, and the rectus muscle which is under examination (the internal, for example) is caught between the teeth of fixation forceps made very light, strong and only two or three centimeters long. A thread extends from the forceps to the upper arm of what is practically a vertical lever. When the person under examination is told to contract the internal rectus muscle by looking in, he draws on the forceps. This makes a pull on the thread and that, drawing on one end of the lever, causes the other end to pass over a graduated scale. If this lever and scale are properly constructed a given amount of motion of the eye indicates that the internal rectus with the other adductors lifts a given number of grams or fractions of a gram. (An illustration of this instrument is here given.)

It is not contended that the data furnished by this appliance are in every way reliable. On the contrary, much still remains to be learned concerning the lifting power of the horizontal recti and the methods of measuring it. But apparently such measurements do furnish at least corroborative evidence of considerable value.

Testimony on this point might also be obtained from one or two other sources, possibly such, for example, as the sound made by the ocular muscles in their contraction, as has been described by Professor Hering, of Leipsic. But these data are apparently of such doubtful value or so very impracticable as to be hardly worthy of mention.

In reviewing, therefore, the different means by which we may learn whether the deviation of an eye in a given case is caused by excessive action of one set of muscles or by insufficient action of another set, it is probable that we must lay most stress on the condition of the refraction, next on the evidence furnished by the perimeter with the tropometer, then the amount of the lifting power, and finally the extent and character of the arc of rotation as shown by the photographic tracings.

But it must be observed that in spite of the most careful tests, frequently repeated, it is practically impossible, in a considerable number of cases, to determine whether an eye deviates in a certain direction because of excessive action of one group of muscles or by insufficient action of the opposing group. It is very certain, however, that the more we study these cases the more do we find to learn concerning them and the more accurate and clear-cut do our ideas become in a given case. It is also certain that there is no inflexible rule for always making tenotomy or for always making advancement. Practice according to such an inflexible rule is hardly less than malpractice. The all-important question is how to select these cases properly. That can only be done by an expenditure of time and patience. It is to offer some slight suggestions as to how this problem can be solved that I venture to present these few points concerning the decision in a given case.

183 Delaware Avenue.

MEMORANDA

OPERATIVE TREATMENT OF PAPILLEDEMA DEPENDENT ON INCREASED INTRACRANIAL TENSION *

G. E. de SCHWEINITZ, M.D.

AND

T. B. HOLLOWAY, M.D.

PHILADELPHIA

INTRODUCTION, WITH HISTORICAL REMARKS

Inasmuch as this paper is concerned only with the operative treatment of papilledema in so far as these operations have included a removal of part of the skull, all reference to lumbar puncture, incision of the distended intervaginal space, etc. is omitted. While it would be interesting to review the earlier operations for the removal of brain tumors, we must content ourselves by referring those who are interested to the papers of Weir and Seguin,[1] P. C. Knapp,[2] and M. Allen Starr,[3] and to those of Knapp,[4] and Spiller and Frazier,[5] for the literature relating to palliative measures.

Although Philip Knapp[6] believes that to Weir should be given the credit of first suggesting the propriety of trephining for the purpose of relieving symptoms where a new growth was inaccessible, or seat could not be determined, it is evident that Sir Victor Horsley,[7] at least three years before Weir's suggestion made at the

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[1] Am. Jour. Med. Sc., 1888, xcvi, 219.

[2] The Pathology, Diagnosis and Treatment of Intracranial Growths, 1891.

[3] Med. Rec., ii, 1, 1896; The Present Status of Brain Surgery, The Journal A. M. A., Sept. 22, 1906, xlvii, 926.

[4] Boston Med. and Surg. Jour., October, 1899.

[5] Univ. Penn. Med. Bull., 1906, xix, 146.

[6] Jour. Nerv. and Ment. Dis., 1892, xvii, 97.

[7] Tr. Tenth Internat. Cong., Berlin, 1890.

first Congress of American Physicians and Surgeons in 1888, had operated for the relief of intense headache and vomiting, and had observed that his operations were followed by an arrest of optic neuritis, a results, as he expresses it, "which in many cases is a source of much amelioration to the patient from the consequent recovery of sight." To this distinguished surgeon, therefore, the fullest credit should be given for this important advance in the treatment of optic neuritis, or choked disc, although in all earlier cranial work, to use a general term, the names of Annandale, Lister, Weir, Horsley, Sahli, Macewen, Jaboulay, Caton and Paul, and Bramwell will always be remembered.

So far as our own country is concerned, it is worth while to make brief reference to some of the early American case histories of patients on whom operation was resorted to solely for the relief of intracranial pressure and headache and occasionally choked disc. Thus, Knapp[8] states that "Horsley's six cases (these being the cases to which Sir Victor Horsley refers in his Berlin address) and the one about to be reported are, so far as I can learn, the only cases in which relief of pressure was the primary object." His patient was operated on in January, 1891, by E. N. Bradford and lived for seven weeks after a trephining which failed to reveal a new growth, but which relieved the patient's pain. Park[9] briefly refers to a patient with inaccessible brain tumor who submitted to operation solely for the relief of increased intracranial tension, and Diller in the same year reported the case history of a patient on whom Dr. Stewart operated for the same purpose. Two years later Keen[10] recorded the case history of a patient on whom he had operated in March, 1892, for an irremovable brain tumor, but with the happy result of relieving the patient of his pressure symptoms, notably headache and hallucinations. In like manner we might refer to the cases of Kammerer.[11] Bartlett and Van Lennep,[12] Diller and Buchanan,[13] Starr,[14] Kramer,[15]

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[8] Jour. Nerv. and Ment. Dis., 1892. xvii. 97.

[9] Med. News. 1892. lxi. 617.

[10] Am. Jour. Med. Sc., 1894, cvii, 138.

[11] Ann. Surg., 1894, xix, 685.

[12] Hahnemann, Month., 1894. new series, xvi, 223.

[13] Med. Rec., 1895, xlvii, 361.

[14] Med. Rec., New York, Feb. 1, 1896. The patient's headache was relieved, but there was no effect on the optic neuritis. The operation was performed by the late Dr. McCosh.

[15] Cincinnati Lancet-Clinic, 1897, new series, xxxix, 523.

Patrick,[16] and Steele.[17] Patrick's case is particularly interesting because it is stated that his patient was trephined for the relief of headache and papilledema. Unfortunately, while the headache disappeared, there was no return of vision, which has been practically lost by atrophy of the optic nerves prior to the operation. The same is true of the case of M. Allen Starr,[14] So, also, is it interesting to recall the sentence of W. N. Bullard,[18] namely, "The operation for the relief of intracranial tension in cases of optic neuritis of unknown origin, apparently not organic, has been performed for me once with excellent success as regards the neuritis. The question to be determined is what chance of recovery the patient has without operation."

No record of this kind would be complete without reference to Harvey Cushing's admirable work, particularly his description of the establishment of cerebral hernia as a decompressive measure for inaccessible tumors and of intermuscular methods of making the bone defect in the temporal and occipital regions.[19] Of great interest in this connection also are his papers on "Experimental and Clinical Observations Concerning the States of Intracranial Tension," which constitute the Mütter Lecture for 1901.[20]

As we have stated in a previous paper,[21] our own attention was first called to this subject by Dr. William G. Spiller at a meeting of the Neurological Society in 1901, and since that date our interest in the matter has been especially stimulated by the opportunity of studying cases in the service of Dr. Charles K. Mills and Dr. W. G. Spiller, and the excellent surgical work of Dr. Charles H. Frazier along these lines in the University Hospital.

In the consideration of this subject we may assume that certain facts are definitely establishel: First, to use the language of Sir Victor Horsley, the release of intracranial tension arrests and cures optic neuritis (papilledema). This has been known in practical work for more than twenty years and has been proved over and over again by surgical experience abroad and in this country.

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[16] Internat. Clin., 1897, series 6, iv, 157.

[17] Chicago Clinic, 1900, xiii, 1.

[18] Intracranial Pressure, Relief in Non-traumatic Cases without Localizing Symptoms, THE JOURNAL A. M. A., 1900, xxxiv, 1658.

[19] Surg. Gynec., Obst. Jour., 1905, i, 297.

[20] Am. Jour. Med. Sc., 1902, cxxiv, 375; 1903, cxxv, 1017.

[21] The Operative Treatment of Papilledema (Choked Disc). With Special Reference to Decompressive Trephining, Tr. Coll. Phys., Philadelphia, 1908, Series 3, xxx, 144

Second, the intraocular changes to which the name "choked disc," or more recently "papilledema" is given, depend in large measure on the effect of increased intracranial pressure. That this is the only etiologic factor may with propriety be disputed; indeed, it seems certain that a combination of factors must be active in the production of this condition. But increased intracranial pressure is the one of which we have most certain knowledge.[22]

Third, the terms "optic neuritis," "descending neuritis" and "papillitis," inasmuch as they give rise to erroneous impressions, or at least describe unproved theories, should be abandoned, and for the same reason "tumor papillitis" and "brain tumor optic neuritis" are not advisable terms. It is unimportant whether we adhere to the old term "choked disc," which Cushing still prefers to all others, or adopt Elschnig's term "papilledema," and we shall consider in the present communication that the two are synonymous.

In the present communication, which we desire to be considered as an elaboration of one we have already made,[21] we have concerned ourselves solely with the results of series of operations—radical in the sense that an intracranial cyst or growth was removed, exploratory in the sense that a craniectomy was performed, sometimes associated with tapping of the ventricles, but no neoplasm was located or removed, and decompressive, i. e., either subtemporal or subtentorial, in the sense that the procedure was undertaken for the sole purpose of releasing increased intracranial tension. We make no reference to operations performed for the relief of epilepsy, etc. without the presence of choked disc, to the removal of epidural and subdural clots, to traumatisms of the skull, to neuritis dependent on constitutional disease, or to lumbar puncture. Moreover, we have described these operations and their results only in so far as they have affected the vision of the patient and the intraocular changes in the nerve-head. For this purpose we have analyzed (1) the literature of the subject since 1900, in so far as it is reported in American medical journals and as it is concerned only with American work; and (2) the cases which have come under our immediate observation in the University Hospital, the Orthopedic Hospital and the Philadelphia General Hospital, of Philadelphia. These investigations bring us into possession of 212 operations on the brain, radical, exploratory or decompressive. Of these 212 cases, 51 have been personally studied by ourselves in the Philadelphia hospitals to which reference has been made, and represent only such cases in which one or other of the operations referred to were performed, and in which the ocular examinations showed the presence of changes in the intraocular end of the optic nerve. The remaining 161 cases have been gathered from the American literature. It has not seemed worth while to add to this analysis literature gathered from abroad, largely because this has been the subject recently of the most thorough investigation, especially by Leslie Paton,[23] von Krüdener,[24] and von Hippel.[25]

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[22] It would be interesting to review, discuss and analyze the various theories which have been advanced to explain the development of "choked disc,. or "papilledema." Time, however, does not permit. Moreover, they have been so frequently and so elaborately presented that it is not necessary. In this connection the reader is referred to The Pathology of the Eye, by J. Herbert Parsons, 1908, iv, 1349-1364; Notes of One Hundred Cases of Intracranial Tumor, With Special Reference to the Ocular Symptoms, by A. W. Ormond, Med.-Chir. Tr., 1906, lxxxix, 489-516; Observations on Choked Disc, with Especial Reference to Decompressive Cranial Operations, by James Bordley, Jr., and Harvey Cushing, Tr. Sect. on Ophth., A.M.A., 1908, 510; Untersuchungen über die Entstehung der Stauungspapille, by Walther Thorner, Arch. f. Ophth., 1909, lxix, 392. The last-named author concludes, after an elaborate review of the literature and as the result of experiments, that among the various theories of choked disc the toxin-theory of Leber best explains the condition. Our own experimental work in connection with Dr. C. H. Frazier, Dr. Edwin Sweet and Dr. F. Prince, Jr., which will be published later, will show, we think, that increased intracranial pressure is not the only factor, even if it is an important one.

Of much interest in this connection is the paper of Harvey Cushing and James Bordley, Jr., Observations on Experimentally Induced Choked Disc, Johns Hopkins Hosp. Bull., April, 1909. Their work, they conclude, "speaks strongly in favor of a mechanical as opposed to a chemical or inflammatory origin for the lesion."

Of the 161 cases just referred to, 29 represent radical operations, 20 exploratory operations, and 13 decompressive operations. Of the remaining 99 operations, some radical and some exploratory and a few decompressive, the accounts are so imperfect as they apply to the visual phenomena, either before or after the opera-

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[23] Tr. Ophth Soc. U. Kingdom, xxv, 1905, 129; 1908, xxvii, 112.

[24] Arch. f. Ophth., 1907, lxv.

[25] Ber. ü. d., xxxv. Versamml. d. deutsch. Ophth. Gesellsch. in Heidelberg, August, 1908.

TABLE 1.—RADICAL OPERATIONS

Case.	Fundus.	Vision Before Operation.	Vision After Operation.	Remarks.
1	No change.	O.D. and O.S., not given; (probably good).	O.D. and O.S., poor (atrophy developed after operation).	Death seventeen months after operation.
2	Choked disc. O.D. 2 mm.; O.S. 2.5 mm.	O.D., 12/XXIV; O.S., 20/XIX.	O.D. and O.S., good sight.	Living and working thirteen months after operation.
3	No change.	O.D. and O.S., not given.	O.D. and O.S., probably good (fundus normal).	At work seven months after operation.
4	Bilateral optic neuritis, 4 D.	O.D. and O.S., not given.	O.D. and O.S., blind.	Relief of general symptoms seventeen months after operation.
5	Double choked discs.	O.D., fingers at 6 inches; O.S., blind.	O.D. and O.S., patient can read some.	General health excellent; no date.
6	O.D. optic neuritis, O.S. beginning optic neuritis.	O.D. and O.S., normal.	O.D. and O.S., preserved at normal.	Death twenty-six months after operation.
7	Double choked discs.	O.D. and O.S., could read.	O.D. and O.S., continued able to read.	Back at work one month after operation.
8	Double choked discs.	O.D. and O.S., "blind."	O.D. and O.S., could read and tell time on watch; discs white.	At work three years after operation.
9	Optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., good sight.	Able to do light work; no date.
10	Double choked disc, 4 D.	O.D. and O.S., not given.	O.D. and O.S., almost blind; discs less swollen.	Death eleven months after operation.
11	Choked discs.	O.D. and O.S., not given.	O.D. and O.S., not given (neuritis returned; no atrophy).	Did well one year; then symptoms returned.
12	Bilateral optic neuritis.	O.D. and O.S., vision much reduced.	O.D. and O.S., regained sight.	Death ten weeks after operation.
13	Choked disc. O.D. 4-6 D.; O.S. 2 D.	O.D. and O.S., hand movements.	O.D. and O.S., recognizes colors; reads newspaper headlines.	Last record eighteen days after operation.
14	Beginning optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., vision better.	Relief of headaches; no date.
15	Double choked discs.	O.D. and O.S., ½-inch type at 15 inches.	O.D. and O.S., rapid improvement in sight.	In excellent condition four months after operation.
16	No change.	O.D. and O.S., not given.	O.D. and O.S., not given (fundus normal).	Last record, five months after operation.
17	Intense optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., vision worse; optic neuritis subsided.	Recovery with blindness; no dates.
18	Bilateral optic neuritis.	O.D. and O.S., counts fingers.	O.D. and O.S., sight improved (end of third day).	Death eleven days after operation.

19	O.D. neuroretinitis, O.S. receding papillitis.	O.D., 20/XXX; O.S., 20/XX.	O.D. and O.S., 20/XV; O.S., disc slightly hazy; O.S., normal.	At work two years after operation.
20	Consecutive atrophy.	O.D. and O.S., not given.	O.D. and O.S., vision improved.	Good result four weeks after operation.
21	Choked discs, with atrophy.	O.D. and O.S., not given.	O.D. and O.S., gradual improvement in sight.	Good; no dates.
22	Choked discs. O.D. 8 D.; O.S. 5 D.	O.D., l. p.; O.S., 20/L.	O.D. and O.S., vision unchanged.	Last record three and one-half months after operation.
23	Beginning optic neuritis.	O.D. and O.S., normal.	O.D. and O.S., not given (discs normal).	Good, eleven months after operation.
24	Double optic neuritis, 2 mm.	O.D. and O.S., not given.	O.D. and O.S., probably good; able to drive wagon.	Able to drive wagon one year after operation.
25	No record; probably atrophy.	O.D., l. p.; O.S., blind.	O.D. and O.S., sees shadows.	Death two weeks after operation.
26	Papillitis.	O.D. and O.S., not given.	O.D. and O.S., not given (disappearance of papillitis).	Good, except continuance of Jacksonian epilepsy.
27	Bilateral optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., saw better; no change in neuritis.	
28	"Pressure neuritis."	O.D., 6/6; O.S., 6/6.	O.D. and O.S., not given (return of neuritis).	Death 108 days after operation.
29	Choked discs with atrophy.	O.D., 7/XX; O.S., 20/XX.	O.D. and O.S., not given (probably good; neuritis disappeared).	
30	Choked discs. O.D. 2-3 D.; O.S. 1½ D.	O.D., 6/6; O.S., 6/5.	O.D., 6/6; O.S., 6/5; O.D., 2 D.; O.S., swelling subsided.	Death one day after radical.
31	Choked discs, 7 D.	O.D. and O.S., not given; (probably blind).	O.D. and O. S., no l. p.; O.D., 4 D.; O.S., 3 D.	Death nine months after operation.
32	Choked disc. O.D. 4 D.; O.S. 3 D.	O.D., 6/9; O.S., 6/7.5.	O.D., 6/9; O.S., 6/9; O.D., 4 D.; O.S., 3 D.	Death one day after third operation.
33	Choked disc. O.D. 5 D.; O.S. 3½ D.	O.D., 6/12; O.S., 6/7.5.	O.D., 6/5; O.S., 6/5; choked disc subsided.	Four months later, reappearance of neuritis.
34	Choked disc. O.D. 5; D.; O.S. 5 D.	O.D., 6/6; O.S., 6/7.5.	O.D., 6/6; O.S., 6/7.5; choked disc subsided.	Reappearance of neuritis and growth several months later. Death after third operation.
35	Choked discs. O.D. 5½ D.; O.S. 5 D.	O.D. and O.S., not given.	O.D., 6/6; O.S., (rapid disappearance of choked disc).	
36	Choked discs. O.D. 8 D.; O.S. 7 D.	O.D., 6/30; O.S., 6/6.	O.D., blind; O.S., 6/30; choked disc subsided, but atrophy.	General condition good, and vision recorded preserved one year after operation.
37	Choked discs. O.D. 5 D.; O.S. 5 D.	O.D. and O.S., 6/30.	O.D., 6/22; O.S., 6/15; disc subsided, 3 D.	Result temporary; two months later, discs atrophic.

tion, that they can not be utilized in the present communication. Of the 51 cases personally investigated, (36 are utilized) of which 8 were radical operations, 7 exploratory operations and 21 decompressive operations[26]; so that, taking all of the cases together, our statistics are as follows: 37 radical operations, 27 exploratory operations, and 34 decompressive operations. Unfortunately, the statistics of Johns Hopkins Hospital, especially as they apply to the work of Dr. Harvey Cushing, were not available. Such cases as have been reported in detail are included in our tables. Dr. Cushing informs us that these statistics in the near future will be brought together and published. We much regret that they could not form part of the present communication, but take this opportunity of expressing our high appreciation of Dr. Cushing's interest in our work and his replies to our inquires, from some of which we shall hereafter quote. We also desire to express our sincere thanks to our colleagues in the University, Orthopedic and Philadelphia General Hospital, Dr. C. H. Frazier, Dr. C. K. Mills, Dr. W. G. Spiller, Dr. W. J. Taylor, Dr. G. G. Davis, Dr. Morris J. Lewis, Dr. Wharton Sinkler, Dr. J. K. Mitchell, Dr. F. X. Dercum, who have permitted us to utilize and examine their cases, to Dr. W. W. Keen, who has furnished us with notes of one important case, and to Dr. H. H. Hoppe, Dr. George Wolsey, Dr. P. C. Knapp, Dr. M. Allen Starr and Dr. J. J. Putnam, who have replied to requests for reprints of articles which they have published on the subject now under consideration.

Most unfortunately, many of the earlier cases have been published without the inclusion of careful ophthalmoscopic and other ocular examination, such examinations as we have endeavored to make of patients in the University and Orthopedic Hospitals, and which we know have been made in the Johns Hopkins Hospital.

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[26] Of the radical operations, 6 were performed by Dr. Charles H. Frazier in the University Hospital and 2 by Dr. W. J. Taylor in the Orthopedic Hospital. Of the 7 exploratory operations, 5 were performed by Dr. Charles H. Frazier in the University Hospital and one each by Dr. W. J. Taylor and Dr. G. G. Davis in the Orthopedic Hospital. Of the 21 decompressive operations, 17 were performed by Dr. Frazier in the University Hospital, one by Dr. Edward Martin in the University Hospital, one by Dr. Keen in the Jefferson Hospital, one by Dr. Taylor in the Orthopedic Hospital, and one by Dr. Joseph Hearn in the Philadelphia General Hospital.

Only too often a case has been reported, either without an ocular and visual examination prior to or after the operation, or with one so imperfectly made that it is valueless in our compilation. We would urge in future work along these lines that the most searching examinations should be made from the ophthalmoscopic standpoint, as well as the standpoint of other visual examinations, both prior to and after the operation, in order that in future statistics a more exact series of conclusions can be reached.

I. TABLES OF OPERATIONS, WITH THE RESULTS OF VISION AND THE OPTIC NERVE-HEAD CHANGES

In order to bring the matter concisely together, we introduce here the tables and the results which have been obtained by the radical, exploratory and decompressive operations.

Summary.—The results in Table 1 may be summarized as follows:

Subsidence of the choked disc and improvement in vision	16
Preservation of vision	6
Primary improvement but later failure or return of choked disc	4
Primary improvement of vision, but early death	3
Unimproved	8
	37

Summary.—The results in Table 2 may be summarized as follows:

Subsidence of choked disc and improvement in vision	7
Preservation of vision	2
Primary improvement but later failure or return of choked disc	2
Primary improvement in vision, but early death	5
Unimproved	2
Early death	9
	27

Summary.—The results in Table 3 may be summarized as follows:

Subsidence of the choked disc and improvement in vision	21
Preservation of vision	5
Unimproved	8
	34

Of the cases marked unimproved in the last table, death occurred comparatively early in 3, namely, at

TABLE 2.—EXPLORATORY OPERATIONS*

Case.	Fundus.	Vision Before Operation.	Vision After Operation.	Remarks.
1	Choked disc, 6-7 D.	O.D. and O.S., blind.	O.D. and O.S., sees shadows.	Death one month after operation.
2	Choked disc.	O.D. and O.S., blind.	O.D. and O.S., not given (choked disc disappeared).	Death one month after operation.
3	Double optic neuritis and beginning atrophy.	O.D. and O.S., not given.	O.D. and O.S., vision improved; less swelling.	After exploratory, a radical. No final statement of results.
4	Choked discs, 5 D.	O.D. and O.S., blind.	O.D. and O.S., no improvement; disc atrophic.	
5	Double optic neuritis.	O.D. and O.S., 20/XL.	O.D. and O.S., no statement except choked disc disappeared three weeks after first operation.	Three operations; death three weeks after third one.
6	Probably subsiding choked disc.	O.D., nearly blind; O.S., blind.	O.D., 5/6; O.S., 3/6, discs pale.	Good result seventeen months after operation; "plays as other boys do."
7	Marked optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., not given; (subsidence of swelling of discs).	Two operations; death eight months after last one.
8	Optic neuritis.	O.D., good vision; O.S., poor vision.	O.D., 20/XX; O.S., counts fingers; O.D., no neuritis; O.S., disc pale.	Two operations; result quoted five months after last one.
9	Optic neuritis.	O.D. and O.S., 6/24.	O.D. and O.S, same vision.	Last report five months after operation.
10	Double optic neuritis.	O.D. and O.S., counts fingers.	O.D. and O.S., primary improvement; later failure.	Three operations; death two months after third.
11	Double choked discs.	O.D., fingers at 2½ feet; O.S., fingers at 3½ feet.	O.D., and O.S., not given (neuritis diminished).	Death one month after operation.
12	Double optic neuritis.	O.D. and O.S., not given.	O.D. and O.S., sees better than before operation.	"Complete recovery."
13	Optic neuritis.	O.D. and O.S., not given.	O.D., blind; O.S., V = "limited to 5 feet."	Death three weeks after operation.
14	No fundus condition noted.	O.D. and O.S., not given.	O.D. and O.S., not given; fundus congested.	Death; no date.

15	Optic neuritis passing to atrophy.	O.D., blind; O.S., "only a little vision."	O.D. and O.S., could distinguish night from day.	No further report.
16	No fundus condition noted.	O.D. and O.S., vision rapidly failing.	O.D. and O.S., eyesight entirely satisfactory.	Ultimate result sixteen months after operation satisfactory.
17	Double choked discs.	O.D. and O.S., vision diminished.	O.D. and O.S., reads print, which was unable to do before operation.	Doing well five years after operation.
18	Neuro-retinitis.	O.D. and O.S., not given.	O.D. and O.S., temporary improvement; later blind.	Last report three months after third operation.
19	Double papillitis.	O.D., 6/12; O.S., 6/12.	O.D., 6/7.5; O.S., 6/7.5; papillitis rapidly subsided.	Death five months after operation.
20	Double choked discs.	O.D. and O.S., not given.	O.D. and O.S., not given; choked disc subsided.	Death three weeks after operation.
21	Choked discs. O.D. 4 D.; O.S. 5½ D.	O.D. and O.S., not given.	O.D. and O.S., no change; O.D., no change; O.S., subsided 1.5 D.	Two operations; death six days after second.
22	Choked discs. O.D. 4 D.; O.S. 2 D.	O.D. and O.S., 6/9.	O.D., 6/60; O.S., 6/60; O.D. +2 D, O.S. +1 D.	Death twelve days after operation.
23	Choked discs. O.D. 2 D.; O.S. 2 D.	O.D. 6/12; O.S., 6/7.5.	O.D. and O.S., not given.	Death one day after operation.
24	Choked discs. O.D. 7 D.; O.S. 6 D.	O.D., 12/L; O.S., 12/CC.	O.D. and O.S., not given (reduction of discs).	Death; no date.
25	Choked discs. O.D. 3 D.; O.S. 5 D.	O.D., 6/12; O.S., 6/15.	O.D. and O.S., no improvement; (increase of disc).	Two operations; the second radical and death two months later.
26	O.D. partial atrophy, O.S. beginning neuritis.	O.D. and O.S., not given.	O.D., 4/10; O.S., 4/25.	Left hospital doing well one month after operation.
27	Beginning neuritis.	O.D., 6/9; O.S., 6/6.	O.D. and O.S., not given; moderate neuritis.	Discharged one month later doing well.

* It has often been extremely difficult accurately to classify the type of operation, and we have included among exploratory operations those in which evidently there was not a simple decompression, but it is actually stated that an exploration was made, or that the tumor was not found, although searched for, or when, craniectomy having been performed, tapping of the ventricles was associated with the operative procedure. It is probable that among these operations some may have been included which were not strictly exploratory, but which for lack of definite statement and because they evidently were neither simple decompressions nor radical operations, we have included in this class.

TABLE 3.—DECOMPRESSIVE OPERATIONS

Case.	Fundus.	Vision Before Operation.	Vision After Operation.	Remarks.
1	Discs partially atrophic.	O.D., 20/XL; O.S., blind.	O.D., 20/XX; O.S., blind.	Ultimate result good.
2	No record.	O.D. and O.S., very poor.	O.D. and O.S., blindness subsided.	Able to go to school one year after operation.
3	Marked choked discs.	O.D., 20/C; O.S., 20/L.	O.D., 20/XL; O.S., 20/XXX; choked discs subsided.	
4	Double choked discs. O.D. 5 D; O.S. 3 D.	O.D. and O.S., not given.	O.D. and O.S., no improvement; disc pale and indistinct.	Relief of headache.
5	Choked disc.	O.D., and O.S., failing vision.	O.D. and O.S., reads fine print; choked disc subsided.	Last report three weeks after operation.
6	No record.	O.D. and O.S., not given.	O.D. and O.S., sight preserved.	
7	Choked discs, 9 D.	O.D. and O.S., not given.	O.D. and O.S., vision lost; choked disc subsided.	Death nine months after operation.
8	No record.	O.D. and O.S., not given.	O.D. and O.S., discs became normal.	No further record.
9	Double choked discs, 7 D.	O.D. and O.S., not given.	O.D. and O.S., discs became normal.	No further record.
10	Double choked discs, 9 D.	O.D., 6/15; O.S., 6/15.	O.D., 6/9; O.S., 6/7.5; disc practically normal.	Still under observation.
11	Choked disc. O.D. 7 D.; O.S. 8 D. Extensive retinal edema.	O.D., 6/100; O.S., 6/150.	O.D., 6/9; retinal edema subsided; O.S., 6/15; discs still swollen.	Still under observation.
12	Choked discs. O.D. 6 D.; O.S. 4 D., subsiding.	O.D., 6/12; O.S., no l. p.	O.D., 6/6; O.S., no l. p.; choked discs subsided.	Good six months after operation.
13	Optic neuritis, 3½ D.	O.D., 20/XXX; O.S., 20/XX.	O.D., 20/XL; O.S., 20/XX; discs flat.	Good two years after operation.
14	Choked disc, 7 D.	O.D., 6/9; O.S., 6/9.	O.D., 6/9; O.S., counts fingers O^2 3.5 D.	Good at last report after operation.
15	Subsiding papillitis, O.D. 4 D; O.S. 5 D.	O.D., ½/150; O.S., no l. p.	O.D., ½/50; O.S., no l. p.; discs flat; atrophy.	Result maintained ten months after operation.
16	Choked discs passing into atrophy.	O.D. and O.S., blind.	O.D. and O.S., no l. p.	Last report two months after operation.
17	Choked discs. O.D. 5 D.; O.S. 4 D.	O.D., 6/45; O.S., 6/45.	O.D., 6/45; O.S., 5/15; discs practically flat; some atrophy.	Doing well two years after operation.
18	Choked discs. O.D. 5 D.; O.S. 5 6 D.	O.D. and O.S., not given.	O.D. and O.S., said to be improved; O.D., subsidence of 1 D; O.S., subsidence of [illegible]	Improved five months after operation.

19	Choked discs. O.D. [illegible] D.; O.S. atrophic.	O.D., [illegible]	disc subsided 2 D.; blind; O.S., atrophy.	
20	Subsiding choked discs.	O.D. and O.S., large letters held close to eye.	O.D. and O.S., no improvement in V.; complete subsidence of choked disc.	Relief of general symptoms; no date.
21	Beginning choked discs, 1½ D.	O.D., 6/6; O.S., 6/6.	O.D., 6/7.5; O.S., 6/7.5; complete subsidence of choked discs.	Good as far as is known.
22	O.D. and O.S. 6-7 D.	O.D. and O.S., 5/6.	O.D. and O.S., 5/30; but probably much better; choked disc practically subsided, 1½ D.	Still under observation.
23	Choked discs. O.D. 11 D.; O.S. subsiding, with atrophy.	O.D., 6/9; O.S., shadows.	O.D., 6/9; O.S., counts fingers; complete subsidence of choked discs.	Good result two years after operation.
24	Choked discs. O.D. 4 D.; O.S. 3 D.	O.D., 6/9; O.S., 6/6.	O.D., 6/6, subsided 1 D.; O.S., 6/7.5, subsided 1.5 D.; exudate thinner.	Improvement continues, nine weeks after operation.
25	Choked discs, 4 D.	O.D. and O.S., only large objects.	O.D., 6/9; O.S., 6/6; complete subsidence of choked disc.	Good result maintained five years after operation.
26	Choked discs. O.D. 6 D.; O.S. 7 D.	O.D., l. p.; O.S., blind.	O.D. and O.S., no improvement; O.D., subsided 1 D.; O.S., subsided 4 D.	Death two months after operation.
27	Choked discs. O.D. 6 D.; O.S. 4 D.	O.D., blind; O.S., 1/100.	O.D., blind; O.S., 1/50; subsidence of swelling.	Death seven months after operation.
28	Choked disc. O.D. 3½ D.; O.S. 2½ D.	O.D., 5/30; O.S., 5/30.	O.D., 5/30; O.S., 5/30; no change.	
29	Choked discs, 5 D.	O.D., l. p.; O.S., l. p.	O.D., l. p.; O.S., l. p.	General condition good, one month after operation.
30	Choked discs.	O.D. and O.S., not given.	O.D. and O.S., not given (discs subsided).	
31	Choked discs.	O.D. and O.S., not given.	O.D. and O.S., not given (cessation of blindness).	
32	Choked discs.	O.D. and O.S., not given.	O.D. and O.S., not given (rapid recovery).	Free from pain and vomiting; no date.
33	Choked discs, subsiding. O.D. 5 D.; O.S. 6 D.	O.D., counts fingers, 6 inches; O.S., counts fingers, 12 inches.	O.D., O.S.	General condition good, eight months after operation.
34	Choked discs, 9 D.	O.D., 2/60; O.S., no l. p.	O.D., hand movements; O.D., subsided 7 D.; O.S., no l. p.; O.S., practically complete subsidence.	Last record one month after operation.

two, nine and eighteen months after operation, and in one of the cases marked "improved" death occurred early, that is, seven months after operation.

It would therefore seem that of the 37 radical operations, improvement of preservation of vision was secured in 22, or 59.5 per cent. The remaining 15, or 40.5 per cent., may be counted, for the various reasons already stated, as failures.

Of the 27 exploratory operations, improvement or preservation of vision was secured in 9, or 33.3 per cent. The remaining 18, or 66.7 per cent., may be counted, for the various reasons already stated, as failures.

Of the 34 decompressive operations, improvement or preservation of vision was secured in 26, or 76.5 per cent. The remaining 8, or 23.5 per cent., may be counted, for the various reasons already stated, as failures.

If, now, we add all of these operations together, 98 in number, improvement or preservation of vision was secured in 57, or 58.2 per cent. and failure occurred, for the various reasons already stated, in 41, or 41.8 per cent.*

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* The three summaries which follow are not included in the tables because it was not possible to ascertain from the descriptions whether or not the individual cases had been previously reported *in extenso* and would be therefore part of the material which we have gathered; hence we give them as nearly as possible as they have been reported by the authors, as follows:

Brush (Med. Rec., 1906, lxx, 215) in 1906 reported 70 cases of brain tumor which had come under observation in the Kings County Hospital and the Brooklyn Eye and Ear Hospital. Among these patients, 19 were submitted to surgical procedures, and the general symptoms were improved or cured in 11 and not mentioned in 4. The condition of the optic nerve was improved in 1, unimproved in 1, and not mentioned in 13. The vision was improved in 3, not improved or unchanged in 4, made worse in 1 and not mentioned in 7.

A. E. Codman (Boston Med. and Surg. Jour., 1905, cliii, 74) reports the results in non-traumatic surgery of the brain and spinal cord from the Massachusetts General Hospital as follows:

1. For the removal of a tumor, if possible, if not, to relieve symptoms caused by pressure, notably headache and failure of eyesight, 28 cases, with death within two weeks in 7, no improvement in 11, improvement in 7, not traced 3.

2. To relieve pressure symptoms; that is, headache or failure of eyesight, 8 cases, with death within two weeks in 4, no improvement in 0, improvement in 2, not traced 2.

There were also 21 operations for the relief of epilepsy and 6 for drainage of a cerebral abscess, but they do not concern us in the present communication.

F. B. Lund (Boston Med. and Surg. Jour., 1905, cliii, 81) makes a report on the operations for cerebral tumor at the Boston City Hospital, referring to 7 cases, although only 6 are quoted in the tables. Apparently in 2 only was there any improvement, and the case records do not make definite statements with reference to the effect of the operations on the visual phenomna.

II. THE OPHTHALMOSCOPIC APPEARANCES OF THE NERVE-HEAD WHICH INDICATE OPERATIVE INTERFERENCE

While in a certain sense the various stages into which systematic writers have divided papilledema, or choked disc, are artificial, they are convenient for descriptive purposes and more accurate than the mere statement that the disc changes were studied at an early or late period of their existence. In our previous paper we employed, with some modifications, the descriptions of Mr. Marcus Gunn,[27] and they are here repeated, again slightly changed.

1. Increased redness of the disc, with blurring of its upper and lower margins, with a gradual progression of the blurring to the nasal edges, while the temporal margin is still visible, represents the first stage.
2. Increased edema of the nerve-head, beginning filling in of the physiologic pit, involvement of the temporal margin of the disc, with a tendency of the edema to spread into the surrounding retinal area, and uneven distention and darkening of the retinal veins, represent the second stage.
3. Decided increase of edema, elevation and size of the nerve-head, with vascular striation of the swollen tissue and striae of edema in the form of lines in the swollen retina between the disc and macula, marked distention of the retinal veins and retinal hemorrhages, represent the third stage.
4. Increase in the prominence of the disc, which assumes a mound-shape and begins to lose its reddish and juicy color and to become opaque, exudation in and on the swollen disc and surrounding retina, elaboration of the retinal hemorrhages in size and number, represent the fourth stage.
5. Decided subsidence of the vascularity of the papilledema and increasing pallor, with or without sinking of its prominence, apparently contraction of the retinal arteries and thickening of their perivascular lymph-sheaths, spots of degeneration of the retina, especially in the macula, represent the fifth stage, which passes into the final stage of so-called papillitic atrophy.

The average swelling of papilledema in cerebral tumors, according to Leslie Paton, is 4 1/2 D. The average swelling of the choked disc, associated with tumor of the brain which we have carefully measured in 19 patients, was in the right eye 4.57 D. and in the left eye 4.63 D. A similar investigation of 24 cases of cerebellar growth (all measurements having been made by ourselves) gave the average height of the right disc as 4.54 D. and of the left disc as 4.63 D. Generally, however, the average height of the discs in cerebellar tumors is greater (from 1 to 1 1/2 D.) than that in cerebral tumors although this difference does not occur in the present series.

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[27] Brit. Med. Jour., Oct. 26, 1907.

In order to gain an accurate estimate of the height of a disc-edema, or of a neuritis, as the case may be, it is evident that at least three measurements should be made, one at the surface of the greatest swelling, one at the macula and one at a position beyond the macula. The conspicuous and often engorged retinal veins, particularly in and on the swelling, should not be taken as points for measurement. We have been accustomed to utilize the vascular striations at the apex of the edematous mound. Mr. Paton suggests that the measurement should be made of the smaller vessels at the most swollen part of the disc. If there is no edema in the macular region, a measurement may there be made of the finer macular vessels, but, as we know and as will be described in the next paragraph, the macular figure is an expression of edema, and if Mr. Paton's figures should be confirmed, in the average number of cases its swelling is even higher than that of the average swelling of the disc-edema in cerebral tumors, 0.6 D. Therefore, to compare the apex of the choked disc with the macula is not safe, but the comparison should be made between the top of the height of the disc and some peripheral portion of the retina, preferably small vessels beyond the macular region. Mr. Paton gives a good illustration of these differences in level. Thus, in one of his cases the apex of the disc was 7 D., the retina beyond the macula 1 1/2 D. and the area of "the macular fan," as he calls it, that is, the area between the disc and the macula, was 4 D. In a recent case under our observation the disc-apex was 7 D., the surface of the retinal edema between 4 and 5 D., and the actual level of the retina 2.50 D.

The macular figures to which reference has been made, and which occur in a fair percentage of cases of cerebral tumor, fully 15 per cent., according to Paton's figures, may begin to develop at that stage in which the severity of the choking of the disc is most marked, therefore in the third and fourth stages. They should be interpreted as indicating an excess of edema, and often are so formed that they proceed gradually from the margin of the disc toward the macular area. In addition to these areas of retinal edema, there may develop at the height of the choked disc in the macular region yellowish-white and degenerated spots intermixed with hemorrhages. Of themselves, according to Bruns, they have serious import and may interfere with vision after the papilledema has disappeared.

It is not infrequently stated that antedating the symptoms which indicate the first stage of papilledema, there may be the so-called "imminence of neuritis or edema," in which the retinal veins, often only one division of them, are darker, more distended and more unevenly tortuous than is normal. It has been suggested that this condition of affairs may be due to pressure on the vein in the intervaginal space, which is already being dilated and which is the place where at the height of the choked disc this flattening of the vessel, as Dupuy-Dutemps has shown, reaches its maximum.[21]

Leslie Paton,[28] however, has not been able to convince himself that venous congestion, as it has been described, precedes the blurring of the disc margins, or that it can be considered as a stage earlier than the so-called first stage of papilledema. Indeed, he is fairly sure that in a number of his cases there was no venous congestion evident to ophthalmoscopic observation when the blurring of the edges was well marked.

While we are satisfied that such venous congestion prior to the blurring of the disc margins is by no means a constant symptom, and as we have elsewhere said, even if it should be manifested, it is not a safe one on which to make a decision as to the propriety of operative interference, we are quite sure that in a few notable instances it has been a conspicuous phenomenon. In one patient whose eyegrounds were under observation for months preceding a radical operation for brain tumor, there was not the slightest ophthalmoscopic change, either in the disc or retinal circulation, until a few weeks prior to his death, when a marked uneven distention of the branches of the upper temporal vein was present for at least a week before the characteristic blurring of the upper and lower disc margins began. This patient was a high myope. In another patient exactly the same phenomena occurred, in this instance the distention being present in both the upper and the lower temporal veins prior to the development of the discedema.

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[28] Ophthalmoscope, March, 1909, p. 146.

Of importance in all cases of increased intracranial tension from the presence of neoplasm is an examination of the visual field, both for form and for colors. Bordley and Cushing have paid especial attention to this matter and their results, which will be recorded in the near future, promise to be interesting. Whether a change in the size of the blind spot, particularly in the vertical direction, which has been found by Ramsay and Sutherland to be an early sign of congestion of the disc in sympathetic ophthalmia, and which depends on turgescence of the superior and inferior branches of the retinal vessels, is present as a sign of early papilledema due to increased intracranial pressure, or, rather, as a sign of vascular congestion when the disc is not yet blurred, we are unable to state. An investigation by us of the size of the blind spot in some cases of papilledema already in the first stage, failed to reveal any change in the long axis of the blind spot.

That an early stage of papilledema may be masked by a corresponding retinal edema, ,is an observation of great interest which has been made by Bordley and Cushing. In one of their cases both disc and retina were uniformly edematous, although the structures remained clear, and it was only after palliative exploration with decompression that the undetected retinal edema subsided in such a manner that the discs at once became prominent, which in their turn subsequently also regained their normal appearance. This observation we can confirm as follows:

Case 1.—A boy, aged 9, with a bullet in his brain, was admitted to the surgical ward in the service of Dr. Frazier in the University Hospital, April 6, 1907, and an ophthalmoscopic examination made three days later records the absence of papilledema, although there appeared to be an ill-defined edema of the retina. On April 13 a decompressing trephining with exploration was performed by Dr. Frazier, and within a

week, with the disappearance of the retinal edema, a marked papilledema, as much as 5 D., was evident. Following a second operation in which the bone was curetted for better drainage, this choked disc, much more marked in the right eye, began to disappear, a month later had decidedly lessened, and we understand at the present time has disappeared, with the preservation of normal vision.

It would therefore seem, as Bordley and Cushing remark, that sometimes, without decompression, an incipient choked disc is extremely hard to distinguish from a simple hyperemia.

It is also difficult to differentiate a true congestion with edema, or beginning papilledema, from a spurious optic neuritis, or so-called hyperopic disc, as it occurs in association with refractive error. There is, however, in the beginning of papilledema, that is to say, when it has reached the first stage, a certain quality which pseudoneuritis does not possess. The blurring of the disc edges is more complete, there is evident contraction or filling in of the physiologic pit, and even with the indirect method of examination, the obscured margins are with difficulty seen, not to mention the uneven tortuosity of the retinal veins, which has already been referred to. A careful investigation of the size of the blind spot and of the light-sense would probably lend aid in diagnosis.

III. THE DATE OF THE OCCURRENCE OF THE NERVE-HEAD CHANGES AND THE FACTORS WHICH DETERMINE THEM

The average frequency of choked disc or papilledema in brain tumor is about 80 per cent., although higher percentages are found in individual series.[29] Usually the intracranial neoplasm must have existed for some time and the increased intracranial tension have lasted for a definite period before the engorgement-edema develops. It is not possible to predict how soon the nerve-head changes will appear after a tumor begins to grow in the brain and the period from the beginning of papilledema to the height of its swelling in some instances comprises only a few weeks; in others, months and even years may elapse before the disc-edema appears. Even tumors of the cerebellum, although they are apt quickly to cause papilledema, do not always maintain this reputation, for, as Bordley and Cushing point out, endotheliomata of the cerebello-pontile angle may exist for a long period of time before they or their results are responsible for a choked disc, in one case as much as eight years. One of us (Dr. de Schweinitz) has discussed this matter elsewhere,[29] and those interested are referred to this paper.

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[29] De Schweinitz, G. E.: The Ocular Symptoms of Tumor of the Cerebrum. Univ. Penn. Med. Bull., April-May, 1906.

Experimental disc-edema, or choked disc following the introduction of fluid into the subdural space, may arise quickly, according to the experiences of some experimenters (Bordley and Cushing,[30] Merz and others). According to others, however, if the fluid used is absolutely bland, not only does disc-edema not develop rapidly, but it is doubtful if it develops at all. A further discussion of this point, however, must be reserved for another communication.

Just as it is not possible to predict how soon after the beginning of a growth in the brain papilledema will appear, so, also, it is not possible to determine with certainty from the stage of the disc or retinal phenomena what the duration of the cerebral lesion is. In the absence of choked disc in brain tumor, there is no means of ascertaining whether or not it will occur. If, however, choked disc arises with great suddenness and the edema rapidly increases, it evidently indicates an increase in intracranial pressure, either because the growth itself has gained in volume, or because hemorrhage has occurred in or around it. As Bordley and Cushing have well said, the character of the intracranial pressure and its effect on the cerebrospinal circulation, regardless of the histology, the situation or the duration of the neoplasm, determines the question which is under discussion.[31]

Choked disc, if it does not fail entirely as a symptom of tumor of the pons, of the medulla and of the corpus callosum, is late in its development, and this lateness of development is also, to a certain extent, true of tumors of the frontal lobe and of the parietal convolutions. Indeed, it is probable that pontine tumors are associated with papilledema, or neuritis, only when they involve some neighboring structure. Paton has summarized his views on this subject in the following sentence: "The great bulk of the cases which do not get optic neuritis are pontine and subcortical tumors; that is, tumors in the white matter of the cerebral hemispheres. If the subcortical tumors spread to the base, then neuritis arises."

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[30] Observations on Experimentally Induced Choked Disc. Johns Hopkins Hosp. Bull., April, 1909.

[31] While Leslie Paton believes that the nature of the tumor apparently has a very small influence in determining the "incidence of neuritis," he modifies this belief by the statement that "in so far as the nature of the tumor may determine the site of the tumor, just so far will it determine the incidence of the neuritis."

We have referred to the severity of the choked disc; that is to say, to the greater edema and swelling of the choked disc in tumors of the cerebellum. According to some authors, the same intensity of the process is evident in tumors of the thalamus and the midbrain. As may be inferred from what has previously been said, it is less pronounced in subcortical, parietal and frontal lobe tumors.

IV. CONCERNING THE VISION OF EYES WITH CHOKED DISC AND ITS RELATION TO THE TIME OF OPERATION

That choked disc caused by intracranial growths is perfectly compatible with good acuteness of vision, and that this may exist for long periods of time, although the edema of the disc-head is well marked, and, finally, that sometimes papilledema may arise, subside, and yet the visual acuteness remain perfect during the whole period of its existence, are well-known facts. As a rule, however, the tendency of choked disc, or optic neuritis, is to cause atrophy of the optic nerve. Hence the earlier that measures are undertaken which dissipate the edema and its results, the better are the chances for the restoration or preservation of vision.

It is important to remember that even where the visual acuteness appears not to be normal it may sometimes be raised nearly or quite to the normal standard if any existing optical defect is corrected. Many examples might be quoted. Two will suffice:

CASE 2.—A boy with moderate optic neuritis following a fracture of the skull was recorded as having a visual acuteness of 6/30, but, after dilatation of his pupil and the correction of 4 D. of hyperopia, his vision promptly rose to 6/9.

CASE 3.—A woman, aged 37, with choked disc of 6 D. and all of the symptoms of cerebellar tumor, had a vision of 6/30,

but with a + 1.50 c axis 180, this vision rose to 6/12, and after the subsidence of the choked disc following a decompressive trephining became 6/6 with the same glass.

Therefore, visual acuteness recorded without note of the refractive error and the effect of neutralizing lenses does not give a true indication of the real sharpness of sight of the eyes with choked disc.

There is much difference of opinion as whether refractive error bears any relationship to the rapidity of the development of papilledema. Mr. Marcus Gunn believes that choked disc is relatively rare in myopia, and it has seemed to us that it does not appear as early, or in as great severity, in myopic as it does in hyperopic eyes. Parsons, on the other hand, doubts if myopia has any deterrent effect on the development of papilledema, an opinion that is shared by Bordley and Cushing. Leslie Paton[32] refers to this matter and thinks that the question is one to which it is difficult to give a definite answer. He has examined six patients with a measured amount of myopia and all had optic neuritis.

Occasionally, as was long ago pointed out by Oppenheim, the choked disc or optic neuritis subsides, although the original disease continues, and, moreover, visual acuteness during this entire period remains comparatively undisturbed. In a recent case under our observation, with no other symptoms except nausea, persistently increasing headache and moderate optic neuritis, the swelling never exceeding 3½ to 4 D., the vision was fully normal in the one and two-thirds of normal in the other eye, and the whole intra-ocular process practically disappeared in the course of several months without the aid of any operative interference. Mercury and iodids were administered. There was not only no improvement in the other symptoms, but, if anything, they grew more severe.

We are accustomed to point out, in discussing radical and palliative operations for the sake of preserving vision, the necessity of early operation, i. e., before the choked disc has passed beyond the second or third stage, and we shall in future pages emphasize this point, but it must be remembered that the ophthalmoscopic appearances alone are not the only guides. In some cases of brain tumor, optic neuritis does not develop; in other cases, before it develops, a not uncommon symptom of cerebral or cerebellar neoplasm may be present, namely, attacks of *temporary amaurosis,* lasting from a few minutes to a few hours, followed by a restoration of the vision to that which existed prior to their occurrence. In the patients whom we have studied, one in particular, the periods of obscuration lasted from a few seconds to six minutes.

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[32] Tr. Ophth. Soc. U. Kingdom, 1908, xxviii, 122.

These amaurotic attacks are the result not of a neuritis or disc-edema, but of compression of the chiasm by the bulged floor of the third ventricle, and, according to Michel, the amaurosis is to be attributed to pressure from the third ventricle, if it appears quickly in both eyes, or rapidly in one eye after the other.[29] One of our patients maintained that sometimes the amaurosis was unilateral, but we could not verify this observation. There was, however, an appreciable difference in time between the development of the attacks and the left eye was the one attacked first and the one in which the blindness lasted longest. The tumor was on the left side of the brain. Evidently if a decompressive trephining for the purpose of saving vision is indicated in the early stage of a papilledema, it is also indicated if the symptoms of increased intracranial tension can be proved to be due to an intracranial neoplasm, even if neuritis or choked disc is not present, but the patient is subject to these attacks of recurring amaurosis due to pressure from the third ventricle. As Mr. Paton puts this matter, it is not necessarily on the first development of the neuritis, but on the first signs of visual failure that palliative trephining should be done with a view to saving vision.

V. THE EFFECTS OF OPERATIVE PROCEDURE, WITH ILLUSTRATIVE CASES

Following the plans pursued in our other communication on this subject,[33] we next record certain propositions and illustrative cases, and wish to point out that we deal only with the effect of operation on eyesight and make no attempt to describe the surgical details.

1. *To Illustrate the Effect of Operation on the Preservation or Restoration of Eyesight:* (A) *when the vision is good in both eyes prior to the operation;* (B)

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[33] Tr. Coll. Phys. Philadelphia, 1908. Series 3, xxx, 144. We shall quote freely from this paper.

when the vision is good in one eye, but practically lost in the other eye prior to operation, and (C) *when the vision is defective; that is below one-half of normal, in both eyes.*

The following cases are illustrative of radical operations under Subdivision A, when the vision is good in both eyes prior to operation:

CASE 4.—A woman, aged 45, under the care of Dr. M. Allen Starr and the late Dr. McCosh[34], with sarcoma of the brain, just prior to operation, has "beginning optic neuritis in each eye," without any apparent effect on vision. A radical operation was performed, the growth having been removed after incision of the brain. One year later the optic discs were normal, the hemiplegia which had followed the operation was almost gone, and the patient had gained twenty pounds.

CASE 5.—A woman, aged 45, with cerebral cyst, had, just prior to the operation, a papilledema of 5 D. in the right eye and 3½ D. in the left eye, and a vision of 6/12 O.D. and 6/7.5 O.S. The patient was admitted to the University Hospital Feb. 5, 1908, in the service of Drs. Mills and Frazier and a large cerebral cyst removed, with complete disappearance of the edema and restoration of full visual acuity at the end of two months. Four months later the edema began again to appear, although vision continued to be normal. When the height of the swelling had reached 5 D., the skull was reopened and a large quantity of growth removed from the site of the original operation, followed, in the course of two months, by complete second disappearance of the papilledema, with a vision of 6/6 or normal in the right eye, and 6/7.5 in the left, on which side there were some indications of pallor in the deeper layers of the disc. Some months after the second operation it became necessary, owing to a return of symptoms, to perform a third operation, which revealed a rapid recurrence of the sarcoma. It was again removed, but a few days later the patient died.[35]

CASE 6.—A woman, aged 23, a patient of Dr. Morris Lewis in the Orthopedic Hospital, with cerebral tumor, had a double choked discs of 5 1/2 D. in each eye, and vision of 6/6. A radical operation was performed by Dr. William J. Taylor over the fissure of Rolando, and the tumor removed. Rapid subsidence of the disc-edema began on the second day and was

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34. Med. Surg. Rep. Presbyterian Hosp., New York, 1902, v, 1.

35. This case is quoted because, although the termination was fatal, it is an excellent illustration of the astonishing effect of technically correct operations in rapidly removing optic neuritis and restoring defective vision, even in the presence of recurrence of a malignant growth. Without these operations the patient would evidently have rapidly lost her vision, and by means of them she had nearly a year of comparative comfort.

nearly complete at the end of the seventeenth day, and the patient is now able to walk around and attend to her affairs. Her vision is normal, and the disc entirely recovered, nearly a year and a half after operation.

CASE 7.—A man, aged 39, under the care of Dr. F. R. Fry and Dr. Mudd,[36] with tumor of the cerebellum, had just prior to operation a vision in O.D of 12/xxiv and in O.S. 20/xix, with double choked discs, the swelling of the right disc being 2 mm. and of the left 2.5 mm. After a radical operation, the tumor having proved to be a conglomerate tubercular neoplasm, the patient did well, and at the expiration of thirteen months was working each day and managing a newspaper, indicating that the sight must have been not only preserved but probably greatly improved.

CASE 8.—A man, aged 56, under the care of Dr. Leszynsky and Dr. J. H. Glass,[37] with tumor of the brain, had prior to operation "neuroretinitis of O.D. and receding papillitis of O.S.," the vision of the right eye being 20/xxx and of the left eye 20/xx. After a radical operation had been performed, the vision rose to 20/xv in each eye, a low myopia having been corrected. The patient made a good recovery, and two years after operation was able to work without headache or any other of the general symptoms which had formerly been present. Partial hemiplegia still remained. The choked discs rapidly disappeared after the operation.

The following case is an exploratory operation in illustration of subdivision A.

CASE 9.—A man, aged 40, under the care of Dr. E. Stieren,[38] had prior to the operation, which was performed by Dr. Stewart, and consisted, after the craniectomy, in an exploration by puncture of the brain without discovery of the growth, well-marked papillitis, his vision with + 2 D. being 6/12 in each eye. After operation the papillitis rapidly subsided, and the vision improved to 6/7.5 in each eye, which vision was retained until the last examination one month prior to the patient's death, which occurred a little less than about five months after operation, autopsy showing that the growth was a vascular glioma.

The following cases present decompressive operations in illustration of subdivision A:

CASE 10.—A girl, aged 14, was admitted to the service of Drs. Spiller and Frazier in the University Hospital on June 4, 1908, with a diagnosis of encephalitis, or possibly of brain tumor, and with beginning papilledema, the swelling of each disc being 1 1/2 D. Vision with test-types was normal in each eye. Decompressing trephining was performed on July 13, 1908, by Dr. Frazier. Six days later, vision still being normal, there was only slight edema of the nerve-heads, and seventeen days after operation there was no swelling of the discs and only the slightest blurring of the nasal margin, vision being practically normal, that is to say, fully 6/7.5 and probably better, as the patient was a little difficult to examine. So far as we are aware, the good result remains at the present time.

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[36] Jour. Nerv. and Mental Dis., 1904.
[37] Med. Rec., 1901, lx, 485.
[38] Ophth. Rec., 1908, xvii, 139.

Case 11.—A man, aged 19, was admitted to the University Hospital on Nov. 6, 1908, under the care of Dr. Edward Martin, and examined neurologically by Drs. Mills and Spiller, who found the evidences of intracranial tumor, probably in the cerebellum. His best vision prior to operation was O.D 6/9, ?, O.S. 6/12 ?, and during a period from the time of admission until Feb. 3, 1909, or about three months, the choked disc of the right eye rose from 5 to 9 D. and of the left eye from 3½ to 9 D., with much retinal edema. Subtentorial decompressing trephining was performed by Drs. Martin, Frazier and Carnett, and within a week there was marked beginning subsidence of the optic neuritis, which at the expiration of six weeks had disappeared, and vision in each eye, which had fallen as low as 6/15 ?, has risen to 6/9 O.D., 6/7.5 O.S.

Case 12.—A man, aged 42, under the care of Dr. Peter Bassoe[39] had symptoms of cerebellar tumor, with a vision of 20/xx in the right and 20/xxx in the left eye, and bilateral optic neuritis, the swelling as measured by Dr. Wilder being 3.5 D. Suboccipital cranial resection with decompression was performed by Dr. A. D. Bevan on Nov. 22, 1906, and almost immediately subsidence of the choked disc began. His severe headaches disappeared, the discs became practically normal, and two years after operation there was no sign of atrophy and the vision of the right eye remained 20/xx, while that of the left eye was 20/xl, or nearly as good as it had been prior to the operation, when it was 20/xxx.

The preceding nine cases, representing five radical, one exploratory and three decompressive operations, illustrate sufficiently the value of operation in the preservation of vision when it is good in both eyes prior to the operation and the papilledema has not passed, at most, beyond the third stage, or, in other words, beyond that stage at which, other things being equal, vision is preserved by a successful operation.

The second subdivision of the proposition is the effect of operation when the vision is good in one eye, but practically lost in the other. This may be illustrated by the following cases:

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[39] Tr. Sect. on Ophth., A. M. A., 1908, p. 545.

Case 13.—A woman, aged 30, under the care of Dr. J. E. Owens of Chicago[40] had a tumor in the right parietal region, with a vision reduced to light perception in the right eye and 20/L in the left eye; choked disc 8 D. right, 5 D. left. While no hope of improving vision was held out to the patient, an operation was performed in two periods, by which the tumor mass was removed, a gold plate having been inserted behind the bone and over the cerebral protuberance. The growth proved to be a round-celled sarcoma, and nearly four months after the operation the patient was in excellent condition, the record being that the vision had not changed. Therefore it was preserved at what it was prior to the operation.

Case 14.—A girl, aged 15, under the care of Drs. J. J. Putnam and Waterman, had prior to operation, which consisted first in exposure of the left lobe of the cerebellum without an opening of the dura, and some days later opening of the dura with the evacuation of a large amount of fluid, although no tumor was found, a vision of the right eye which was nearly normal while with the left eye the vision was very poor, and the patient could recognize with difficulty people whom she knew. There was well-marked optic neuritis. Five months after operation the vision of the right eye was 20/xx, and of the left eye, fingers at one foot. All signs of optic neuritis had disappeared, but the left disc had an atrophic pallor.

Case 15.—A man, aged 20, referred to the University Hospital, Oct. 25, 1905, by Dr. Weisenburg, with cerebellar tumor, had papilledema of the right eye of 11 D. and a vision of 6/9, and subsiding papilledema of the left eye, with a vision of only shadows. Simple decompression was performed by Dr. Frazier without opening of the dura, and the first subsidence in the swelling of the edematous disc of the right eye was noticeable twelve days after operation. At the expiration of two months the papilledema had disappeared and the vision was 6/9 in the right eye, while the vision of the left eye has risen to counting fingers. In this eye there was temporarily an increase of 1 D. in the swelling of the disc, which later slowly subsided. Five months later a second decompressing trephining on the opposite side of the skull was performed by Dr. Frazier because of some increase in the patient's general symptoms, the vision of the right eye remaining 6/9 as before, but the left, which showed increasing atrophy of the disc, had dropped again to shadows. The patient has remained well, and at the last report, two years after operation, he was performing his duties as a working man each day. It can not be doubted that had the decompressive trephining not been performed, the right disc would have met the same fate which befell the left one.

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[40] Med. Recorder, 1905, xxvii, 554.

CASE 16.—A woman, aged 37, under the care of Drs. W. W. Keen and F. X. Dercum, with symptoms of brain tumor but without localizing signs, had an uncorrected vision of 6/30 of the right eye and no light perception in the left eye. The choked disc of the right eye measured 6 D. and of the left eye 4 D. While all the signs of subsiding optic neuritis were present, that in the right eye might have been considered to be in the late third or early fourth stage. The visual field was much contracted. A subtemporal decompression was performed by Dr. Keen on Jan. 8, 1909; at the expiration of the third day subsidence of the choked disc was evident, and at the expiration of three weeks all swelling of the right disc had largely disappeared, although there was still some filling in of the central cup and the veins were full and tortuous. Vision, however, had risen, with proper correction, to 6/6. The vision of the left eye was unchanged by the operation, although the swelling of the disc had somewhat subsided, about 1 D.

Considering the good result in each of these cases, whether by the preservation of the vision which had existed before the operation, or by its actual improvement, it may be safely assumed that had it been possible to operate while vision still remained in the poorer eye of each of these patients, it also would have been conserved. The cases also illustrate the unevenness of the process, in so far as the optic nerves are concerned, and that, even if atrophy has begun on one side, it does not necessarily follow that in the other eye the optic nerve will exhibit an atrophic process, and, therefore, operation is indicated.

The third subdivision of the proposition is the effect of operation when the vision is defective; that is, below one-half of normal in both eyes. To illustrate this the following cases may be quoted:

CASE 17.—A man, aged 23, was admitted to the University Hospital, Dec. 12, 1903, with cerebellar tumor and a papilledema of the right eye of 4 D., and in the left eye slightly less; he had very poor vision, which unfortunately was not recorded with test-types at the time of his admission, but which is reported to have been "only the ability to see large objects." A decompressing operation was performed by Dr. Frazier, with removal of a portion of the cerebellar lobe, and at the expiration of seven days the vision had risen to 6/15 in the right eye and 6/7.5 in the left eye, and the edema of the disc had dropped 1 D. on the right side and 2 D. on the left. At the expiration of eleven weeks after the operation the vision of the right eye had risen to 6/9, and of the left to 6/6. In the right eye there was only a slight surface edema, and the left eye was normal. Five years after operation both discs were normal, and the vision of the right eye was 5/7.5, and the left 5/5.

Case 18.—A patient, aged 40, whose case history has been reported by Dr. G. Robinson,[41] had prior to operation double choked discs, more pronounced on the right side, and a vision of 20/C O.D, 20/L O.S. After a subtentorial decompression by J. T. Schell on the right side, the patient made a rapid recovery, the swollen disc subsided and the retinal edema disappeared, and at the time of the report the vision of the right eye was 20/xl and of the left eye 20/xxx.

Case 19.—A man, aged 59, was admitted to the University Hospital Jan. 28, 1909, in the service of Dr. Spiller and Dr. Charles H. Frazier with double choked discs, the point of highest edema in the right eye being 8 D. and in the left eye 7 D. There was marked edema of the retina between the discs and the macula, although there was no macular fan, and the edematous area was 5 D. The retina beyond this, that is, beyond the macula, was 2 D. Loalizing symptoms were not present, but a subtemporal decompression was performed by Dr. Frazier on Jan. 21, 1909, followed by the escape of an unusual amount of cerebrospinal fluid. Convalescence was uninterrupted, and the edema of the retina began to subside at the expiration of twenty-four hours. At the end of two weeks, when he left the hospital, the vision of the right eye, which prior to the operation had been 6/100, was 6/9, and that of the left eye, which had been 6/150, was 6/15, with suitable correction. The discs, although less swollen, were still edematous, in the right eye 6 D. as compared with 8 D. prior to the operation, and in the left eye 6 D. as compared with 7 D. The retinal edema, however, had entirely subsided. The patient is still under observation. There was also a marked improvement in the field of vision, which prior to the operation had been greatly contracted and exhibited large central scotomas on the nasal side.

These cases illustrate the advantage of operation even when the vision prior to its performance is not encouraging. Evidently the process has not yet reached the stage of destruction of the function of the nerve fibers, and with the release of intracranial pressure the subsidence of the edema and its results took place, followed by improvement in vision.

All of the sixteen cases are good examples of the fact, now so well and frequently established, that with the relief of intracranial tension by operation, provided the choked disc has not passed beyond the third stage, the prognosis as to vision is decidedly favorable.

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[41] Pennsylvania Med. Jour., 1909, xii, 327, and Ophthalmology, January, 1909.

2. *To Illustrate the Effect of Operation When Vision Is Very Poor and the Choked Disc Has Passed Beyond the Third Stage Prior to Its Performance.*—Unfortunately, many patients come under observation long after the favorable time for operating has passed in so far as a restoration of vision is concerned. Hence it becomes important to examine the records as to the value of operation in its relation to the restoration or preservation of vision in these late stages. The question to be answered is this: Shall the surgeon operate, even though the papilledema has reached the fourth or even the fifth stage? Certainly even in the fourth stage the prognosis *quoad visum* after operation is unfavorable, and so high an authority as Mr. Gunn states that if the fifth stage is reached there is no reason, so far as sight is concerned, for performing any operation, but he is willing that the operation shall be performed in the fourth stage, because he has known useful vision to be obtained after operation even at this period.

We shall divide the illustrative cases as follows:

A. *The Restoration of Useful Vision After a Late (Fourth) Stage of Papilledema Is Present.*

CASE 20.—A woman, aged 40, under the care of Dr. J. J. Putnam and Dr. G. A. Waterman[42] had prior to operation choked discs in a late stage, with vision of O.D counting fingers at 6 inches, O.S. "blind." A round-celled sarcoma was removed by Dr. Mixter from the left lateral lobe of the cerebellum. As soon as vision could be tested after operation the patient appeared to be blind, but nineteen months later "he could read some" and used his eyes more than before the operation. At this date the general health was excellent, and he was able to walk alone, although he still had some weakness of the left side.

CASE 21.—A man, aged 18, under the care of Dr. Hoppe[43] had prior to operation well-marked subsiding choked discs. He is stated to have been blind and the blindness was preceded by left hemianopsia. A glioma the size of a German walnut was removed from the left occipital lobe. Vision steadily improved, and some time after the operation he "could read headlines in a paper and tell the time on a watch." Three to four years after the operation the record is that the patient could see well.

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[42] Jour. Nerv. and Ment. Dis., 1906, xxxiii, 297.

[43] Hoppe, H. H.: A Report of Seven Operations for Brain Tumors and Cysts, THE JOURNAL A. M. A., Feb. 2, 1901, xxxvi, 302.

CASE 22.—A woman, aged 35, under the care of Dr. H. A. Bruce,[44] had prior to operation double choked discs, O.D 4—6 D., O.S. 2 D., which appeared to be subsiding. Vision was reduced to hand movements. Operation revealed a cyst in the left cerebellum, which was evacuated Nov. 14, 1906. Nov. 29, 1906, the patient could recognize colors and read the headlines of newspapers, and the last record, namely, Dec. 2, 1906, states that the eyesight was steadily improving.

CASE 23.—A boy, aged 10, under the care of Dr. J. J. Putnam and Dr. Waterman[42] had prior to operation a vision as follows: O.D. nearly blind, O.S. blind. Operation was performed April 29, 1904, by Dr. Mixter in two stages. Exploration failed to find a tumor. Five months later the patient read with difficulty with O.S. but readily with O.D., and the final record of vision, Sept. 15, 1905, or seventeen months after the operation, is O.D. 5/6, O.S. 3/6; disc of O.S. very pale and sharply defined, O.S. similar but less marked.

CASE 24.—A child (age not given), under the care of Dr. M. Allen Starr,[45] was operated on by Dr. Cushing by means of a decompressing trephining over both lobes of the cerebellum. There is no statement as to the condition of the eyes prior to operation, but the comment is that "blindness has subsided," from whence it may be inferred that the vision prior to operation was very poor, and the ultimate result is contained in the following statement: "Although he staggers somewhat in walking, he lives in comfort and is able to go to school."

Decidedly, the results of such operations are encouraging, and if the chance of restoring to a patient who is practically blind enough vision to read newspaper headlines, or to tell time on a watch, is presented, he should have the full benefit of it.

B. *The Preservation of, or Even Slight Improvement in, Very Poor Vision as a Result of a Late Stage of the Papilledema.*

CASE 25.—M..B., a woman, aged 29, with ceerbral tumor, had papilledema in the fourth stage of 3 D. in the right eye and 4 D. in the left eye, in which, indeed, the signs of the fifth stage were beginning to be manifest. The vision of the right eye was ½/150; the left eye was totally blind. A decompressive trephining was performed by Dr. Frazier, and at the expiration of ten months after the operation, at which time the last ocular examination was made, the vision of the right eye had improved from ½/150 to ½/50; all swelling of the discs had long since disappeared.

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[44] Annals of Surg., 1907, xlv, 543.

[45] The Present Status of Brain Surgery, THE JOURNAL A. M. A., Sept. 22, 1906, xlviii, 926.

CASE 26.—A boy, aged 15, with cerebellar tumor, blind in the right eye and with a vision of 1/100 in the left eye, had double papilledema in the late fourth of beginning fifth stage of 6 D. in the right eye and 4 D. in the left eye. Decompressive trephining, with opening of the dura, was performed by Dr. Frazier, and the first signs of subsidence of the choked disc were manifest on the nineteenth day, and at the expiration of two months almost all of the swelling had disappeared, and the vision of the left, or only eye which had any sight prior to the operation, was still preserved, in fact was slightly better, being 1/60 as compared with 1/100.

Certainly these two cases are good illustrations of our contention, and also the contention of Mr. Gunn, that, even though the visual prognosis is unfavorable, and a late stage of the papilledema has been reached, blindness may still be averted and a decompressive trephining should be tried.

C. *The Effect of Operation in Preserving the Light Perception, Which Was the Only Vision Existing Prior to Its Performance.*

CASE 27.—A man, aged 19, under the care of Dr. W. Leszynsky,[46] had prior to operation intense papillitis, 6 to 7 D. in each eye, and vision is recorded as "blind." Operation was performed on June 1, 1903, by the late Dr. McCosh. Exploration failed to find a tumor in the region of the Rolandic fissure. After operation the patient seemed to have better vision. He could see shadows and describe the style of dress worn by nurses. Death occurred five weeks after operation, and the autopsy revealed a soft red mass in the right ventricle, which appeared to spring from the inner wall of the ventricle. It was thought to be a glioma.

CASE 28.—A girl, aged 14, with double papilledema of 5 D. in a late stage of its development and vision reduced to faint light perception, had a decompressive trephining with opening of the dura performed by Dr. Frazier. Already at the end of the first day there was distinct subsidence of the swelling, and one month later it had absolutely disappeared, fortunately leaving the patient with the light perception that existed prior to the operation, but without improvement.

All who have to do with the blind will realize how much it means to the sufferer if he has only light perception, and how he will cling to that small blessing with almost the same tenacity with which we should cling to the preservation of good vision. Therefore, it would seem that, even from this standpoint, it is fair to give the patient a chance by a decompressive trephining, although there is no hope of restoring vision, but only hope of preserving this faint light perception.

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[46] Jour. Nerv. and Mental Dis., 1904; also Med. Rec., 1904, p. 165. In the first journal the patient is stated to have been a woman, in the second a man.

The pathetic side of such records is the knowledge that, had these patients been able to submit to operation at the proper time, there is reason to believe that sight would have been preserved.

D. *The Apparent Effect of Operation in Hastening the Loss of Vision When This Has Been Greatly Reduced Prior to Its Performance by a Late Stage of the Optic Nerve Changes.*

It should be remembered, however, that in a certain number of patients, the subjects of brain tumor who have very little vision prior to operation, for example, a vision reduced to hand movements, or even light perception only, this sight may rapidly disappear after the trephining. For example, to quote from our previous paper, three patients, one with a tumor of the cerebellum, another with a growth at the cerebello-pontile angle, and a third with a cyst of the left lobe of the cerebellum and with much reduced vision prior to operation, namely, hand movements in two and the ability to distinguish very large letters held close to the eye in a third, were completely blind, even to the abolition of light perception, within the first week after operation, although in each instance the surgical success was a brilliant one. Such experiences, however, should not be regarded as contraindications for the operation, because these patients with very much reduced vision are doomed to speedy blindness without operation, while with it at least they have a small chance and, in any event, if the procedure is a success, they are relieved from distressing general symptoms, headache, nausea, vomiting, etc.

VI. CERTAIN COMPLICATIONS AND DISAPPOINTMENTS CONNECTED WITH THE OPERATION

1. *Little or No Subsidence of the Choked Disc, but Marked Improvement in Vision.*—All those who have interested themselves in this subject must have observed occasionally that, although the operation is not

followed by any marked subsidence in the choked disc, visual acuteness greatly improves. For example:

Case 29.[47]—A patient came under observation in the University Hospital while under the care of Drs. Spiller and Frazier, whose choked disc on the right side was 7 D. in the upper part of the swelling, and 8 D. in the lower part of the edematous disc, which was particularly spread out downward and outward; while on the left side the swelling was more marked below and to the nasal side, where it reached 7 D. The retina between the disc and the macula was edematous, thrown into folds, although there were no lines of white edema, and the height of these folds reached in places 5 D. The refraction of his eyes was as follows:

O.D. + 2.50 $\overset{\frown}{\smile}$ +.50 c, axis 75.
O.S. + 2.

Vision, O.D 6/100, O. S. 6/150. The visual field was irregularly contracted and there were large scotomas almost symmetrically placed in the nasal half of each field of vision. After a decompressive trephining performed by Dr. Frazier, the vision began rapidly to improve, and at the end of one day had risen in the right eye to 10/cxcii and in the left eye to 10/cxxi (test-types of the American Ophthalmological Society). There was practically no change in the swelling of the discs, but the retinal edema was beginning to subside. At the expiration of nine days the vision of the right eye was 6/15, of the left 6/30; also no distinct subsidence in the choked discs. Seven days later the vision of the right eye was 6/9 and of the left eye 6/15, with a loss of 2 D. in the swelling of the right disc and 1 D. in the swelling of the left disc. The retinal edema, however, had entirely disappeared.

Doubtless in cases of this character there is ultimately a disappearance of the papilledema and the primary improvement in visual acuity before the choked disc disappears is due, in the first place, to restoration of the edematous retina to its normal plane, and, in the second place, to the relief of pressure from the distended third ventricle.

2. *The Occasional Delay in the Subsidence of the Papilledema and the Restoration of Vision, with an Ultimate Good Result.*

Case 30.—A woman, aged 51, with a tumor of the cerebellopontile angle, had a papilledema of 4 D. in the right eye and 3 D. in the left eye and a vision of 6/9 in the right and 6/6 in the left. After decompressive trephining with opening of

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[47] This case is also referred to under another heading. See case 19.

the dura, there was absolutely no change in the appearance of the discs at the expiration of several weeks. After a second operation performed by Dr. Frazier, in which the dura was opened and some blood evacuated and the dura left unsewed, there was again no change for nearly a month, but on the twenty-third day the disc-edema began to subside, and five weeks later the swelling of the discs had been reduced to 2 D. in the one and to 1 1/2 D. in the other eye, and vision had improved in the right eye from 6/9 to 6/6, and was practically maintained in the left eye at 6/7.5.

Cases of this kind illustrate the accuracy of Mr. Gunn's statement, that "visual improvement after operating, although long delayed, may ultimately be very satisfactory."

3. *Increase in Papilledema and Visual Disturbance Following Decompressive Trephining, Radical, or Exploratory Operation.*—In a certain number of patients a temporary depression of vision within the first week after operation is demonstrable. Exactly to what this is due is not clear. Possibly it may be attributed to hemorrhage, perhaps to shock, but in any event it apparently is of no importance in the subquent restoration or preservation of vision, provided those changes which lead to atrophy of the optic nerve fibers have not already begun. Slight increase in edema, sometimes associated with fresh hemorrhages in and around the disc, is another phenomenon not infrequently observed after these operations, which disappears in the subsequent general subsidence of the swelling. For example:

Case 31.—A woman, aged 17, with sarcoma of the brain and double papilledema of 5 D. each, had a decompressive trephining performed by Dr. Frazier, with opening of the dura. At the end of eleven days vision had decreased and the papilledema increased 2 D. on the right side and 1 D. on the left, but one month later there was gradual improvement, followed by the usual subsidence.

Such temporary increase of edema may be due, as has been pointed out by Bordley and Cushing and other observers, to manipulation of the brain at the time of operation, or may depend on hemorrhage which has gathered beneath the dura and which must be evacuated at a subsequent operation. Under such circumstances, therefore, the prognosis is usually not unfavorable, and the increase in edema should cause the surgeon to make a careful examination of the field of operation, to ascertain whether hemorrhage or accumulated serum pent up beneath the dura should be evacuated.

4. *The Development of Hemorrhages in and Around the Disc, or in Distant Portions of the Retina.*—As is well known, the third and fourth stages of papilledema may be associated with hemorrhages, especially situated in various shapes and sizes around the margin of the swelling. Such hemorrhages Paton attributes not to the severity of the neuritis, but to "a particular condition of the blood or the blood vessels in the patient under observation."

The occasional increase in edema with a few fresh hemorrhages after operation has been referred to, but in some patients after palliative trephining or radical operation a decided development of fresh hemorrhages near the disc or in distant parts of the retina is evident. In the cases we have studied this has been due to degenerative changes in the retinal vessels, and was not caused directly by the operation. An illustrative case is the following:

Case 32.—Tumor of the cerebrum; double choked discs, right + 4 D., left + 5.50 D. Removal of the tumor by Dr. Frazier in two operations four days apart. Five days later increased neuritis and many fresh hemorrhages near the swollen nerve-head and scattered over the retinas; distinct sclerosis of the retinal vessels; general arteriosclerosis.

5. *Preservation of the Vision of One Eye, but Failure to Preserve the Sight of the Opposite Eye by Operation.*—An occasional disappointment in the result of operation is its failure to exercise its beneficial effect in equal degree on both eyes, although the vision of each was equally good and the papilledema apparently not beyond the favorable stage. For example:

Case 33.—A woman, aged 21, with cerebral tumor, had a vision of 6/9 in each eye and a papilledema in the third stage of 7 D. in each eye. A decompressive trephining, with opening of the dura, was performed by Dr. Frazier; at the expiration of nine days there was marked subsidence of the swelling, and after one month it had been reduced just 50 per cent., with complete preservation of the vision of the right eye, but with a reduction of the vision of the left eye from 6/9 to counting fingers.

In such cases it will be found, we think, that the eye which fails to retain or regain its vision is homolateral to the tumor, and probably the process, although not yet ophthalmoscopically evident, has extended to such a degree that, in spite of the subsidence of the edema, atrophy of the nerve fibers pressed on by new-formed elements takes place. The mere degree of the swelling of the papilla evidently is not always an accurate indication of the visual prognosis, and even the ophthalmoscopic examination does not invariably reveal satisfactorily whether the earlier stages of atrophy have begun. A careful visual field and color and light-sense tests are necessary to complete the evidence.

6. *Loss of Vision Which Has Been Good Prior to the Operation and with the Papilledema Still in an Early Stage.*—Occasionally, after technically correct operations, when one would expect good results, there is rapid loss of vision. That this may occur in patients in whom prior to the time of operation there has been decided depreciation of vision has already been described, but there are a number of cases on record in which a precisely similar untoward result develops, although the vision may be good, sometimes only in one eye and at other times in both eyes. The most notable example in our series is the following:

CASE 34.—A boy, aged 10, a patient of Dr. Sinkler's in the Orthopedic Hospital, with cerebellar cyst and double papilledema of 8 D. in the right eye and 7 D. in the left eye, had a vision of 6/30 in the right eye and 6/6 in the left eye. Trephining was performed by Dr. William J. Taylor, and the operation suspended on account of severe hemorrhage. At the expiration of thirteen days there was not the slightest change in the swelling of the discs. Twenty days after the first operation a second one was performed, the dura opened, and the cyst ruptured during the manipulation, with the result that at the end of three days the swollen discs had been reduced respectively to 3 and 5 D., and at the expiration of a little more than a month their swelling had entirely disappeared; but in the meantime rapid atrophy had ensued, and the right eye had become totally blind and the vision of the left eye reduced to 6/60 with difficulty. The last examination indicates slight visual improvement, namely, 6/30.

A number of explanations have been offered to account for this accident. One of us (Dr. de Schweinitz) suggested that it might be possible that this unfortunate sequel was due to great loss of blood at the time of the operation, the hemorrhage having caused some change in the ganglion cells of the retina, exactly as it does in some cases of free loss of blood from the stomach or bowels. Dr. Cushing, in a personal communication on this subject, based on an experience of his own, suggests that, although the exploration may have revealed no lesion, the patient may have a growth involving the corpora quadrigemina with implication of the geniculate bodies, and points out also that in cases of cerebellar cyst there may be a gradual shutting down of the nerve of the new-formed connective tissue during the process of subsidence of the swelling.

In conclusion, we would call attention to a few practical points connected with this subject:

1. *The Earliest Date at Which the Subsidence of the Swollen Disc Is Noticeable.*—In this connection we quote from our previous paper as follows:

"In eleven cerebellar tumors recently observed, the first distinct lessening in the disc edema after operation, that is to say, at least 1 D., was noticed as early as the end of the first day in one case and deferred as late as the twenty-third day in another case, the average being a little over nine days. A similar analysis of nine cerebral tumors showed that the first decrease occurred in one case as early as the end of the second day, and in another case, in which there was a primary increase of the edema, it was delayed to the thirtieth day, the average being a little over ten days. A very common result, even in those cases in which the disc edema itself does not appreciably diminish for the first few days, is a lessening of the general vascularity of the eyeground, which is often quite noticeable within a few hours after the trephining, although we have not been able to observe it while watching the eyeground during the progress of the operation. This, however, is rather difficult to do, and doubtless future investigations along these lines may yield other results. In general terms, it may be stated that, while the disc edema may subside as much as 1 D. within the first twenty-four or forty-eight hours, and quite commonly begins to subside from the third to the fourth day, an average of a number of cases usually indicates that the first decided improvement occurs from the ninth to the tenth day. This observation is in accord with the statistics already published by Mr. Paton, who states that generally within a week or a fortnight after the second operation (all of his cases were submitted to two operations, or, rather, to two stages of operation, the first consisting in removal of the bone and sometimes incision of the dura, and the second, if possible, the removal of the tumor itself) there was distinct subsidence of the swelling, but between the two operations there was little measurable alteration in the discs."

According to Mr. Gunn, the date of the first occurrence of the subsidence of disc swelling and the rapidity with which it proceeds vary considerably, but are earlier and more rapid in cases where the operation has been performed not later than the third stage of disc edema. We have not found in our observations that there was any material advantage, in so far as the preservation or restoration of vision is concerned, in sudden subsidence of disc edema, or, more accurately, in rapid subsidence, with perhaps one notable exception, operated on at the Orthopedic Hospital by Dr. Taylor. Indeed, we have come to believe that the more gradual subsidence, beginning at about the tenth day, yielded more favorable results than when the disc elevation subsided more rapidly. This point, however, is one which would require further study before it could be presented in a dogmatic statement.

2. *The Indications Which the Disc Appearances Give as to the Size and Situation of the Growth, That Is to Say, Whether It Is on the Right or the Left Side of the Brain or Cerebellum.*—As is well known, ophthalmoscopic examination yields no satisfactory evidence in regard to the size or structure of a tumor, inasmuch as small growths may cause an elevation of intracranial pressure greater than large and infiltrating ones, depending on the situation of each. What the nature of the tumor itself has to do with determining the "incidence of neuritis," to use Mr. Paton's expression, we have already discussed, and have also recorded this observer's views. Bordley and Cushing call attention to the fact that if the growth has caused an increase in intracranial tension through the intermediation of an internal hydrocephalus, the disc-edema is apt to be equal in the two eyes, and these authors believe that if every brain tumor exerted its pressure equally in all directions the swelling of the two discs would always be likewise equal. This, we know, is not always the case.

Whether the greater swelling on one side is a sufficient indication that the growth is also on the same side of the intracranial contents is much disputed. Sir

Victor Horsley[48] is outspoken in his belief that neuritis (choked disc) tends to develop earlier and to be more marked in the eye corresponding to the side on which the tumor grows, and further believes that certain contradictions of this view are due to the "treachery of clinical statistics." He insists that every case of optic neuritis must be dealt with on its own merits and not classified with others. He further insists that the character of the neuritis must be studied, whether there is exudate, and, if so, how much in each disc; in other words, the minutest detail of each eyeground in each case must be examined and recorded, and he deprecates very much the publication of statistics which tend to eliminate what he considers to be an invaluable clinical sign, namely, the homolaterality of the neuritis. This view was shared by Sir William Gowers and the late Dr. Beevor, as well as other observers.

Mr. Paton, on the other hand, doubts if reliance can be placed on this sign, for, although the preponderance in his investigations was somewhat in favor of the more severe neuritis being on the affected side, yet in some cases it was more marked on the opposite side. In his most recent communication on this subject,[49] including only such cases which have been watched through the various stages of the neuritis and in which there were no signs of commencing subsidence or atrophy in one eye or the other, he gives the results of 102 records. In 53 the difference in swelling between the two eyes did not amount to more than 0.5 of a diopter, that is to say, the swelling was practically the same in the two eyes. In 25 the swelling was greater in the eye corresponding to the side of the tumor, while in 24 the swelling was greater in the contralateral eye. In a definite number of the cases the neuritis developed while the patients were under observation, and in 13 of them the neuritis commenced practically simultaneously in the two eyes. In 11 the swelling began first in the contralateral eye, and in 6 it developed first in the homolateral eye. Moreover, in 4 of the cases the neuritis appeared only in one eye, and in each of these 4 it was the contralateral eye which showed the neuritis. With a record such as this, he is naturally opposed to the placing of any reliance on this clinical sign.

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48. Tr. Ophth. Soc. U. Kingdom, 1908, xxviii, 136.
49. Ophthalmoscope, March 1, 1909, p. 148.

Bordley and Cushing state that in 70 per cent. of their cases the papilla on the side of the tumor was the most involved, and in 80 per cent. of the cases seen before the choked disc made its appearance the swelling ultimately occurred in the eye homolateral to the tumor, and in 72 per cent. the ultimate damage to the nerve was greatest on this side.[50]

J. M. Martin,[51] in his well-known analysis of the reports of 601 brain tumors, endeavors to show that the seat of the lesion is probably on the side on which the optic neuritis is more marked in the proportion of 71 to 29.

In our previous communication on this subject we stated that, in our own experience, in the majority of cases, the greater swelling was on the same side as the tumor, but a more careful examination yields the following data: The swelling was practically equal in the two eyes in 33.3 per cent. of the cases; was greater in the eye on the same side as the tumor in 46.6 per cent. of the cases, and was greater in the eye on the side opposite to the tumor in 20 per cent. of the cases. The number of cases examined, however, is too small to render these statitstics of much value. Moreover, it was not possible to submit them to the requirements on which Mr. Paton very properly insists.

We are in entire accord with Sir Victor Horsley, Harvey Cushing and others, that it is not so much to swelling of the disc that attention should be paid as to the indications which disc has been longest affected by finding the signs of beginning atrophy, by attention to which side of the disc has been first affected, and by tests of the visual acuteness, the light-sense and the field of vision, and if these precautions are taken we are satisfied that ocular examination is of the greatest value in guiding the surgeon to the side on which the skull should be opened.

3. *Delay in the Development of the Optic Disc Changes.*—The delay in the development of papilledema, depending on the size, situation and character of the growth, has already been discussed, as well as the relation of refractive error in this respect. Certain tables published by Singer[52] bear on the point at present un-

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[50] Tr. Sect. on Ophth., A. M. A., 1908, p. 510.

[51] Lancet, London, 1897, ii, 83.

[52] Lancet, London, 1902, ii, 1687.

der discussion, because, if he is correct, the absence of optic neuritis or disc-edema in intracranial tumors, exclusive of those occurring in the pons, is rare in those under 40 years of age and becomes increasingly more frequent after that period of life. Thus, in an analysis of 88 cases of cerebrum and cerebellar tumor, he found in 35 patients under 30 years of age well-marked choked disc in 97.1 per cent. of the cases, and its late or slight development in only 2.9 per cent. of the cases, it being absent in none of them. Between 30 and 40 the figures read thus: Eighteen cases well-marked neuritis, 76.6 per cent.; late or slight neuritis, 2.2 per cent.; absent, 1.1 per cent.; between 40 and 50 the neuritis was well marked in 61.9 per cent. of the cases, 21 in all, and absent in 19; while over 60 years of age in 7 cases it was not well marked in any of them, late or slight in 14.3 per cent. of them, and absent in 57.1 per cent.

4. *Certain Therapeutic Considerations.*—While in a certain number of operations performed for the relief of choked disc and other associated symptoms of intracranial pressure a good result has followed when the dura has not been opened, it is, we believe, the universal opinion of surgeons and neurologists that this structure should be divided after the bony portion of the skull has been removed, because without such division a true decompression is not accomplished. It is foreign to our purpose to discuss the technic of operation, which will be described by Dr. Frazier, and, therefore, we content ourselves with this single statement.

A matter of importance, however, is the administration of remedies after operation. Only too frequently valuable time has been lost by the administration for prolonged periods of time of iodids and mercurials in the hope of relieving the patient's general and ocular symptoms, but after operation there is no reason why these remedies should not be exhibited, and in point of fact it has seemed to us in a number of cases that the ultimate good result was hastened by their employment. It stands to reason that if there is any efficacy in the use of alterative remedies, such efficacy will be materially increased after the reduction of the increased intracranial tension by the operation. We have, therefore, been accustomed to suggest to the physicians and surgeons in charge of the cases which we have had the opportunity of studying that, provided there were no contraindications and as soon as feasible after the operation, iodids and mercurials should be administered, and sometimes we have combined with the iodid the bromid of potassium and Fowler's solution. We have also thought it worth while to exhibit, especially in patients of advanced years in whom there were indications of general arteriosclerosis, evident either by ophthalmoscopic examination, or suggested by investigation with a sphygmomanometer, small, frequently repeated doses of nitroglycerin.

Finally, we will quote and emphasize the conclusions which we reached in our previous paper, namely:

1. The most satisfactory treatment for the purpose of preserving vision in any case of choked disc or papilledema not due to a toxic process or constitutional disease (infectious optic neuritis), but depending on increased intracranial tension, is decompressive trephining, with the removal of the growth if it is accessible.

"2. This operation should be performed early, and, if it can be done during the first, second or even the third stage of papilledema, the prognosis as to sight is most favorable.

"3. If for any reason the operation is postponed until the development of the fourth and fifth stages of papilledema, already associated with marked depreciation of vision, the prognosis as to sight is unfavorable, but even under these circumstances the operation should be performed, because it sometimes preserves such vision as still remains.

"4. In non-syphilitic cases, time devoted to the administration of iodids and mercurials is time wasted, but after the operation their exhibition appears to exert a beneficial influence.

"5. The investigation of the eyes must include not only an ophthalmoscopic examination, but also a careful estimation of the visual field, the color perception, the light-sense and the size of the blind spot.

"6. Patients afflicted with papilledema depending on increased intracranial tension should have the case fairly stated to them, and the operation should be urged, in spite of the occasional unfavorable results, because in its absence ultimate blindness is almost always sure to result."

1705 Walnut Street—1928 Chestnut Street.

MEMORANDA

THE SURGICAL ASPECTS OF CEREBRAL DECOMPRESSION

WITH REMARKS ON THE ETIOLOGY AND CERTAIN MANIFESTATIONS OF PAPILLEDEMA *

CHARLES H. FRAZIER, M.D.
Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University Hospital
PHILADELPHIA

Though having had the privlege of presenting a paper on a kindred subject to this Section but a year ago, your Secretary has been kind enough to invite me to participate in your transactions again and has assigned to me the topic of the "Surgical Aspects of Cerebral Decompression." Rather than to any contribution of my own, I attribute this opportunity chiefly to the fact that one of your members, Dr. de Schweinitz, has been so closely identified with all the neurologic cases in my clinic, and in this connection I should like to emphasize the importance not only from the standpoint of the patient's welfare, but as a means of accurate and reliable observation of the closest coöperation, between the ophthalmic and neurologic staff on the one hand and the surgical on the other. An exhaustive study of the case before operation at the hands of the experienced neurologist and ophthalmologist, their observations of the lesion at the time of the operation, and their continued studies of the after-effects serve as a safeguard against surgical blunders and guarantee accuracy and reliability in the published records.

Of all the phases of papilledema, none has aroused more interest and discussion than the etiology. The conditions in which it has been associated are well

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known, notably tumor, hydrocephalus, and more recently traumatic lesions. It has been assumed by some that the most important factor in its pathogenesis is increased intracranial tension. Plausible as this theory is, it is not borne out by experimental evidence, at least with one or possibly two exceptions experimentors have thus far failed to produce a papilledema solely by increasing intracranial tension. Because of the existing obscurity as to its etiology, I have with the collaboration of Drs. de Schweinitz, Sweet, Prime and Holloway, made a series of observations in the Laboratory of Experimental Surgery of the University of Pennsylvania, for the purpose of determining for ourselves whether increased tension did or did not play any part. The experiments will be published in detail together with others bearing on the subject of decompression so that I shall allude here only to the essential features.

Two methods of increasing intracranial tension were employed, the wet and the dry. By the dry method, a small rubber bag was introduced beneath the skull after splitting the dura and retained in place by a special appliance. By elevating the reservoir, the bag was distended with water to the point of maximum tolerance. By the wet method, normal saline solution was introduced through a small canula screwed into the skull; the dura was opened and the rate of flow was regulated by elevating or lowering the reservoir. In some cases, the fluid was introduced into the pretentorial space, in some into the subtentorial space. The degree of intracranial tension was determined by kymographic tracings of the blood pressure, the respiration and in some instances of the brain pressure. Examination of the eyegrounds were made by Dr. de Schweinitz or by Dr. Holloway, before the experiment was begun and afterward at intervals of every few minutes. The degree of intracranial tension was recognized by the increase in blood pressure and by the partial or complete arrest of the respiratory function. In every experiment, tension was increased up to a point where the respiratory function was arrested and it became necessary to resort to artificial respiration to resuscitate the animal.

The following is the summary of the experimental observations:

Case 1.—Summary: Dry method. Extreme degree intracranial tension; no evidence of choked disc; slight dilation and pulsation of nasal branch of retinal veins. The changes in the vessels were quite as marked on the opposite side as on that on which pressure was inserted.

Case 2.—Summary: Dry method. Under a degree of intracranial tension, sufficient to cause arrest of respiratory function and elevation of the blood pressure, signs of intracranial tension continued from one to two hours. There were no changes in the eyeground other than filling and subsequently pulsation of the retinal veins.

Case 3.—Summary: Dry method. Increase of intracranial pressure attended with most profound disturbance of the respiratory and circulatory centers caused practically no changes whatsoever in the eyeground, not even the filling and pulsating of veins as observed in previous experiments.

Case 4.—Summary: Wet method. Intracranial pressure increased by introduction of 185 c.c. of normal saline solution in subtentorial region. Much smaller quantity required to cause changes in retinal vessels and the death of the animal than in pretentorial region. No signs of papilledema.

Case 5.—Summary: Wet method. The enormous amount of saline solution (2,520 c.c.) which was absorbed in so short a time (two hours) in itself was remarkable. The eye symptoms, except exophthalmus, were not particularly marked; no edema or pulsation having occurred. At the autopsy all the organs, with the exception of the lungs, were filled with fluid.

Case 6.—Summary: Dry method. Increase of intracranial pressure (by distention of a bag inserted within the cranium) up to and beyond the physiologic limit, as indicated by the arrest of the respiratory and circulatory function, failed to provoke papilledema. At times the discs were bleached; at times the veins were distended, and at times there was pulsation of the vessels, but nothing more.

Case 7.—Summary: Wet and dry method combined. The intracranial pressure was increased with the distended bag until there were signs of intracranial tension; the latter was increased up to and beyond the maximum degree of toleration by the introduction of 100 c.c. of normal saline salt solution. No changes in the eyegrounds were observed.

From these experiments the following conclusions are drawn:

1. Increase of intracranial tension by artificial means up to a point attended with profound disturbance of the circulatory and respiratory functions was in no instance attended with development of a papilledema, except in two instances not here recorded, and in these there was

no marked swelling of the disc, but only a slight edema and filling in of the center of the discs.

2. The duration of the observation varied from two to ten hours; in one instance increased intracranial tension was maintained and observations made from six o'clock in the evening to four o'clock the next morning. So that the element of time did not affect the results.

3. In some instances the eyegrounds were bleached (the stage of anemia) and in many there was more or less fulness and occasionally pulsation of the retinal veins, but nothing else.

4. The degree of intracranial tension artificially produced in these experiments exceeded by far that which obtains in the vast majority of these lesions of which papilledema is a clinical manifestation.

5. Therefore, in the etiology of papilledema some factor or factors other than increased intracranial tension play an important part.

6. What these may be is still a matter of conjecture.

Papilledema and its relation to intracranial trauma is a subject which has commanded our attention for a number of years. The statement has been made that papilledema is a more or less constant accompaniment of those cases of intracranial trauma in which there is obstruction to the venous circulation, the stage of *Stauungs hyperemic* which, according to Kocher's scheme, is the first stage of cerebral compression. These include cases of cerebral contusion, in which edema is a conspicuous feature, although it might include also cases of large intracranial hemorrhages. For the past two years Dr. de Schweinitz has examined almost all the cases of this description in my service at the University Hospital, and in all he has discovered but two cases of papilledema. This is an insignificant number on which to base any conclusion, but a small number of carefully studied and observed cases is often of greater value than a mass of imperfectly digested material.

Because of their direct bearing on not only the etiology of papilledema but on the question of operative interference for relief, I will refer to these cases in some detail.

CASE 1.—A young man, aged 25, sustained an injury to his head in falling from a trolley car.[1] There was no positive evidence of fractrue of the skull and the case was regarded as one of cerebral contusion. The principal symptoms were vomiting, delirium and frontal headache. The latter was of intense and persisted in this intense form for at least ten days after the accident. The pupils were equally dilated and there were no focal symptoms. The superficial veins of the scalp were conspicuously dilated.

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[1] Index File No. 4259. For the report of this case I am indebted to Dr. Edward Martin.

January 10 (seventh day): Dr. de Schweinitz examined the eyes with the following result: The right disc edematous; + 3.5 D.; veins enormously distended; retinal hemorrhage below disc; left disc + 5 D.; veins distended and hemorrhages above and below disc.

January 13 (tenth day): Swelling of discs not so marked between 3 and 4 D. Veins not so full. Vision O. D. 4/5 O. S. 4/6.

January 19 (sixteenth day): R. O. highest surface of disc + 4 D.; some hemorrhage L. O. disc + 4 O.; hemorrhage above; veins very full.

The patient was discharged from the hospital but returned to the out-patient clinic for further observation. Within a few weeks the vision was entirely normal and the papilledema had entirely disappeared.

In this case we see a type of intracranial trauma which would be regarded as a concussion or, more properly, a contusion without evidence of fracture but with symptoms persisting for almost two weeks, at no time attended with unconsciousness, stupor or convulsions, but with a moderately high grade papilledema which subsided spontaneously, that is, without operative intervention. The question of a decompressive operation was considered, but because of the absence of conditions that could be said to be alarming, was not resorted to.

CASE 2.—A woman, aged 35 years[2] fell from a trolley car and sustained a serious injury to her head. On admission she was completely unconscious and did not respond to any external stimuli. There was a large hematoma on the left occipitotemporal region, but no fracture could be felt. There was no bleeding from the nose, mouth or ears; the pulse was slow and intermittent and the blood pressure 128 millimeters of mercury. Examination of the urine discovered 3.5 per cent. of sugar. There were no sensory and no motor phenomena except in the muscles supplied by some of the cranial nerves. There was some weakness of the muscles supplied by the left facial nerve. In examining the eye, Dr. de Schweinitz reported as follows: R. O. oculomotor palsy; superior, inferior and internal rectus involved; ptosis not quite complete; pupils dilated about five millimeters; no reaction to light; well

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[2] M. B. Index File 4362.

marked edema of the disc + 3 D.; enormous dilation of the retinal veins and moderate tortuosity; arteries unchanged; L. O. no involvement of the external ocular muscles; condition of the disc and retinal veins same as on the other side. The symptoms were attributed to a serious cerebral contusion and the involvement of the cranial nerves to small nasal hemorrhages, possibly to a fracture of the petrous bone.

January 10 (seventh day): All the edges of the discs were clear except the nasal; the veins were distinctly smaller; right eye still diverges.

January 20 (seventeenth day): The edges of both discs were perfectly clear and the veins not far from normal in size.

The patient gradually recovered consciousness and from the subsequent mental confusion, and as her life was at no time threatened operative interference was not contemplated.

Whatever other indications there may be for decompression in cases of intracranial trauma, it would appear from these at least that papilledema alone can not be regarded as one. In both instances the papilledema measuring + 7 and + 3.5 diopters respectively subsided spontaneously within a comparatively short time after the accident, *i. e.*, between two or three weeks.

Furthermore, I wish to call attention to the relative infrequency of papilledema as a manifestation of serious types of intracranial trauma. That it may exist in certain instances there is no doubt, but its comparative infrequency, minimizes its significance as a diagnostic sign and as a means of determining the degree of intracranial tension and the necessity of relieving it by surgical measures.

The question of the relation of the papilledema to the size, situation, duration and nature of the tumor is not a new one. That the size of the tumor has little to do with the degree of elevation of the disc is the concensus of opinion; that a cortical or a basal tumor is more apt to cause papilledema than a subcortical, in the white substance of the brain, is also well known, and finally that tumors in the subtentorial region lead to papilledema earlier in the course of the case than pretentorial cases, will not be questioned. In tabulating a series of 19 cases, selected only from patients examined by de Schweinitz and subsequently operated on by myself, I found that with two exceptions (in which the discs measured + 7 and + 9 diopters respectively) whenever a papilledema was present the measurements were rather uniform; that is to say, the majority

TABLE 1

No.	Name.	File No.	Diagnosis.	Operation.	Eye Examination Before Operation.	Eye Examination After Operation.	Resul .
1	L. E. P.	560	Sarcoma cerebellum.	12, 17, '03. Craniectomy; removal of small tumor.	12, 8, '03. O. D. + 7 D. O. S. + 7 D.	1, 25, '04. O. D. + 4 D. O. S. + 3 D.	Died 10, 27, '04.
2	M.G.J.	1782	Tumor cerebello pontile angle.	10, 15, '06. Unilateral decompression; suboccipital craniectomy.	10, 10, '06. O. D. + 4 D. O. S. + 3 D.	12, 9, '06. O. D. + 3 D. O. S. + 1 5 D.	Still living.
3	F. M...	578	Sarcoma of cerebellum.	3, 13, '04. Suboccipital craniectomy and removal of portion of cerebellum.	3, 7, '04 O. D. + 4 D. O. S. + 3 D.	3, 26, '04. No change.	Recovered; died subsequently.
4	G. E...	697	Tumor of cerebellum.	12, 14, '03. Unilateral suboccipital craniectomy; portion of cerebellar (⅜) hemisphere removed.	12, 12, '03. O. D. + 4 D. O. S. less.	11, 6, '07. Entirely subsided.	Recovered; still living.
5	P. K...	1054	Cerebellar tumor.	5, 17, '05. Unilateral suboccipital craniectomy; portion of hemisphere removed.	5, 12, '05. O. D. + 6 D. O. S. + 4 D.	7, 21, '05. Discs show practically no swelling.	Recovered; died 12, 24, '05.
6	S. L...	1157	Cerebellar tumor.	10, 26, '05. Unilateral suboccipital craniectomy; dura not opened.	10, 23, '05. O. D. + 5 D. O. S. optic atrophy.	2, 1, '06. O. D. + 1 D. O. S. + 1 D.	Recovered; still living.
7	S. L...	1157	Cerebellar tumor.	Second operation 6, 14, '06. Dura split......	6, 11, '06. O. D. + 1 D. O. S. + 1 D.	6, 21, '06. O. D. + 1 D. O. S. + 1 D. Practically flat.	Recovered; siill living.
8	T. H. A.	3107	Sarcoma cerebello pontile angle.	12, 8, '07. Unilateral suboccipital craniectomy.	12, 6, '07. O. D. + ⅔ D. O. S. + 1.5 D.	1, 8, '08. O. D. + 2 D. O. S. Swelling subsided.	Died subsequently, after radical operation.
9	E. F...	1267	Tumor of cerebellum.	3, 10, '06. Unilateral suboccipital craniectomy.	Well-marked atrophy.	Well-marked atrophy.	Recovered; still living.
10	H. M..	3686	Tumor of cerebellum.	6, 29, '08. Bilateral suboccipital craniectomy; dura split.	6, 26, '08. O. D. + 5 D. O. S. + 5 D.	7, 30, '08. O. D. + 1 D. O. S. Atrophy; practically no swelling.	Recovered; still living.
11	M. S...	3697	Tumor of cerebellum.	6, 30, '08. Unilateral (left) craniectomy.....	6, 28, '08. O. D. + 8 D. O. S. + 8 D.	7, 31, '08. O. D. + 1 D. O. S. No swelling.	Recovered; still living.
12	E. D...	1231	Tumor of cerebellum.	2, 26, '06. Unilateral suboccipital craniectomy; dura not split.	2, 15, '06. O. D. + 6 D. O. S. + 7 D.	3, 25, '06. O. D. + 4 D. O. S. + 3 D.	Death.

measured from + 3.5 to + 4 or + 4.5 D. Irrespective of duration, of the nature of the tumor, whether infiltrating or more or less encapsulated, with the size varying from that of a chestnut to a hen's egg, there was a comparatively uniform elevation of + 3 to + 4 diopters. In other words, a trivial and insignificant variation.

If I may be permitted to refer to another threadworn subject, namely, the equality or inequality of the discs in the affected as compared with the unaffected side, I would like to place in evidence a series of cases in which the discs were examined by Dr. de Schweinitz before the operation and we were able at the operation to expose the tumor. In these there was not enough difference between the disc of the affected and that of the unaffected side to enable us to draw any conclusions as to the seat of the tumor. This testimony is in absolute disaccord with the very positive statements of Horsley, whose operative experience has been so exceptionally large, but it agrees with the more recent observations of Mr. Paton. I present this series, small as it is, for what it may be worth, and I may add, I think, without contradiction of Drs. de Schweinitz, Mills, or Spiller, who have examined practically every patient operated on in my clinic, that in cases in which there was any doubt as to whether the lesion was on one side or the other, it was the exception rather than the rule to be able by examining the swelling of the discs alone to draw any definite conclusions. Much help, however, can be obtained by studying the age of the nerve-head changes, that is, which disc has been longest affected, by finding the signs of beginning atrophy, etc., points which Horsley also considers most important.

In dwelling on these various aspects of papilledema I have wandered somewhat afield from the subject assigned me, namely, "The Surgical Aspects of Cerebral Decompression." Reference has already been made to the propriety of decompressing in cases of intracranial trauma with papilledema. Apart from trauma, decompression is practiced as a means of relief in certain cases of hydrocephalus, tumors and internal hydrocephalus, but as there are no ocular manifestations of importance in epilepsy I shall confine my remarks chiefly to tumors with or without hydrocephalus.

TABLE 2

No.	Name.	File No.	Diagnosis.	Operation.	Eye Examination Before Operation.	Eye Examination After Operation.	Result.
1	W. D..	557	Tumor of brain.	11, 7, '03. Decompression.	11, 5, '03. O. D. nerve head swollen; O. S. nerve head swollen.	1. 2, '04. O. D. + 3 D. O. S. + 4 D.	Recovery; still living.
2	J. K...	3116	Tumor of brain.	1, 10, '08. Decompression.	Marked papilledema.......	Papilledema entirely disappeared.	Recovered; still living.
3	P. S...	1268	Tumor of brain.	3, 20, '06. Decompression.	1, 29, '06. O. D. + 5 D. O. S. + 4 D.	11, 6, '07. Papilledema practically disappeared; discs show beginning atrophy.	Recovered; still living.
4	M. B...	2084	Tumor of brain.	1, 13, '07. Decompression.	1, 5, '07. O. D. + 3-4 D. O. S. + 3-5 D.	11, 6, '07. Both eyes advanced optic atrophy; all swelling gone.	Recovered; living 11, 6, '07.
5	I. K...	1937	Tumor of brain.	11, 28, '06. Decompression.	11, 19, '06. O. D. + 5.5 D. O. S. + 5.5 D.	1, 5, '07. O. D. + 2 D. O. S. + 2 D.	Recovered.
6	E. U...	1862	Tumor of brain.	11, 1, '06. Decompression.	10, 26, '06. O. D. + 5 D. O. S. + 5-6 D.	12, 9, '06. O. D. + 4 D. O. S. + 4 D	Recovered; died subsequently.
7	M. B...	802	Hydrocephalus unilateral of brain.	10, 10, '04. Occipital decompression.	10, 3, '04. O. D. + 4 D. O. S. + 4 D.	10, 24, '04. O. D. + 2 D. O. S. + 2 D. 10 weeks after operation choked disc entirely subsided.	Recovered; still living.
8	I. D....	978	Tumor of brain.	6, 12, '05 Suboccipital decompression.	6, 9, '05. O. D. + 8 D. O. S. atrophy.	6, 16, '05. O. D. + 6 D. O. S. atrophy.	Death 2 weeks after operation.

It is a well-known fact that the large majoritty of tumors, whether pretentorial or subtentorial, are inoperable. That is to say, the majority are either inaccessible or impossible of localization, or of such a nature as to make their removal inconsistent with the maintenance of life. To express the percentage of operable tumors in figures is not an easy matter; to estimate it from the results of postmortem observations is obviously misleading; to say that from 75 to 80 per cent. are inoperable is by no means a conservative estimate.

In the surgery of brain tumors there are but two propositions to consider: the radical operation, implying the removal of the tumor, and the palliative operation, cerebral decompression. From what has been said of the operability of brain tumors it will be seen at a glance that in the majority of instances the most we can offer is palliation. But in the case of brain tumor palliation implies much more than it does when applied. to other incurable lesions. That is to say, given a patient with an inoperable brain tumor palliation means restoration or preservation of vision, relief from headache and from persistent vomiting, three symptoms which together combine to make the patient's life utterly wretched and unbearable. Without them she or he may continue to live for years in comparative comfort.

Decompression is a very much less formidable procedure , it can be performed in a much shorter time and with but trifling risk to life. It implies the removal of an area of bone with a diameter of from 3 to 5 cm. from the temporal region or occipital region on one or both sides and a free opening in the dura or the removal of a portion of it. Whether or not to decompress in the temporal region will depend to a great extent on the situation of the lesion. In all pretentorial tumors temporal decompression is the operation of choice and in all subtentorial lesions a suboccipital decompression is indicated. Usually, sufficient relief from intracranial tension will be afforded by a unilateral operation; should the tumor grow rapidly and the symptoms recur the operation may be repeated on the opposite side.

For the purpose of studying the effects of decompression on a papilledema when performed in the temporal and suboccipital regions I have tabulated two series of cases (See Tables 2 and 3) from Dr. de Schweinitz's records. Although the disc examination was not made

Table 3

No.	Name.	File No.	Papilledema.	Duration.	Situation.	Nature.	Approximate Size.
1	L. E. P..	560	O. D. + 7 D. O. S. + 7 D..	10 months...	Left cerebellar hemisphere.............	Sarcoma, sharply defined.	Hazelnut.
2	M. G. J..	1782	O. D. + 4 D. O. S. + 3 D...	2 years......	Left cerebello-pontile angle.............	Fibrosarcoma...	Horse chestnut.
3	T. H. A..	3107	O. D. + 2-. D. O. S. + 1.5 D.	7 years......	Right cerebello pontile angle...........	Sarcoma	Hickorynut.
4	M. S.....	3697	O. D. + 8 D. O. S. + 8 D...	6 months....	Left cerebello pontile angle.............	Unknown.	Hickorynut.
5	E. D.....	1231	O. D + 6 D. O. S. + 7 D...	2 years......	Right cerebello-pontile angle...........	?	Hen's egg.
6	Mrs. S...	542	O. D. + 4 D. O. S. + 5-5.5 D.	1 year.......	Left cerebello-pontile angle.............	Neurofibroma. .	Chestnut.
7	I. D.....	978	O. D. + 8 D. O. S. atrophy.	1½ years.....	Left temporal lobe at base...............	Sarcoma	5×8 cm.
8	A. G.....	836	O. D. + 3 D. O. S. + 5 D..	2 years......	Right parietal region (cortical)..........	Sarcoma	3.5×3 cm.
9	W. D....	3340	Subsiding neuritis; swelling greatest on left side.	18 months...	Left parietal region (cortical)...........	? Glioma........	3.75×2 cm.
10	J. S.....	624	O. D. + 5 D. O. S. + 4 D..	4 years......	Right frontal (cortical)..................	Sarcoma	6.25×6.25 cm.
11	G. A. H..	2146	No papilledema.............	6 months . .	Left parietal (cortical)..................	Sarcoma	Hen's egg
12	A. C. E..	3690	No papilledema.............	....	Left parietal (cortical)..................	Glioma..........	7×6×3.5 cm.
13	G. K.....	464	O. D. no papilledema. O. S. beginning neuritis.	Several years.	Left parietal (cortical)..................	Sarcoma	Small.
14	H. S. G..	1214	No examination.............	5 months....	Right occipital and temporal at base...	Sarcoma	Child's fist.
15	A. K.....	2223	Extensive papilledema. O. D. + 7 D. O. S. + 7 D.	1 year......	Left temporal lobe at base..........	?	Large size.
16	E. U.....	1862	O. D. + 5 D. O. S. + 5-6 D.	6 months....	Right parietal (cortical)................	Sarcoma	Large.
17	M. B.....	802	O. D. + 4 D. O. S. + 4 D...	10 months...	Right occipital (subcortical)............	Cyst.	Capacity 1½ oz.
18	Mrs. J. L.	3205	O. D. + 5 D. O. S. + 3.5 D.	4 months....	Right occipital lobe (cortical)...........	Cyst.	

at the same interval after operation in all cases, it would seem that the results of temporal decompression were about as good as these of suboccipital decompression or *vice versa.* While I have never decompressed in the temporal region for lesions of the cerebellum, it is reasonable to assume that by this method we might not obtain the necessary relief of tension for lesions beneath or behind the tentorium. In the case of tumors the cerebellar hemispheres are usually under much greater tension than the cerebral hemispheres, often so great that nothing but the removal of bone from directly over the seat of the hemisphere would afford adequate relief.

Cerebral decompression is called for, as a rule, in inoperable tumors; it may be resorted to, however, as a matter of temporary expediency, when the conditions do not admit of delay and the tumor can not be localized. Delay is admissible when the papilledema has persisted from six weeks to two months, whether or not there has been temporary improvement under the influence of mercury or the iodides, when procrastination will lead inevitably to optic atrophy and to blindness.

There should be no question about the advisability of operating in these comparatively early cases if only to save vision. The propriety of operating in the advanced cases, when the patient is already in deep stupor, may be questioned. Bruns, for example, opposes such a course. It should be borne in mind that with the patient unconscious the operation can be performed, as I have had occasion to do, without an anesthetic. The operation can be completed in twenty minutes and, while adding but little to the gravity of the situation, may save the patient's life. A striking example of this was brought to my attention in a patient referred to me by Dr. Mills. The case seemed hopeless—the patient was unconscious when brought to the hospital and her life was despaired of. A rapidly executed decompression was followed by subsidence of some of the symptoms with recovery of consciousness. On the other hand, in a similar patient recently operated on for Dr. William G. Spiller, the operation seemed to have no effect on the fatal tendencies.

Finally, a few words as to the immediate and ultimate effects of decompression. At the site of the operation there developes a hernia protruding more or less according to the degree of intracranial tension. This is usually well covered with hair and therefore particularly in women, is not very conspicuous.

The mortality following operations on patients with cerebral or cerebellar tumors will depend on the condition of the patient and the seat of the lesions. The longer the duration of the lesion and the nearer the growth to the pons and medulla the greater the danger. All told, in the past seven years I have had only two fatalities following temporal decompression for cerebral tumors. In one of these the patient was stuperous when operated on, and at the autopsy a large tumor was found extending from the temporal lobe back to and including the pons. In the other the tumor also involved the pons. Of the suboccipital decompressions for tumors or suspected tumors of the posterior fossa in the same period there have been five fatalities, one in each of the years from 1904 to 1908. But three of these cases were operative deaths in the strict sense; that is, the patient never fully recovered from the shock of the operation. The two remaining lingered several weeks.

Vomiting and headache, one or the other or both, are relieved in about two out of every three cases by decompression.

In a word, the effects of decompression are in the main beneficial; in fact, so beneficial that we should not question the propriety of operating even though the case may at first seem hopeless. The only grave and fortunately a very rare consequence is sudden blindness. This has never occurred in our service, and apart from this I have never seen any aggravation of the condition of more than a transitory character.

The expectancy of life is a matter of no little interest; even though the patient survives the ordeal but a year, it is not a little in this period to have saved sight, allayed vomiting and relieved headache. In exceptional instances a longer lease on life may be anticipated. Among my patients is one operated on in 1902, one in 1903 and two in 1904. The larger the tumor and the nearer its approach to the vital centers the shorter will be the period of survival.

MEMORANDA

THE PROPHYLAXIS OF INTERSTITIAL KERATITIS*

H. GIFFORD, M.D.
Professor of Ophthalmology and Otology in the University of Nebraska
OMAHA

Considering the great interest to the oculist of interstitial keratitis, the question of its prophylaxis has received very shabby treatment. While it may be discussed in some of the numerous works that I have not seen, I have found no mention of it in any of the dozen English, German, and French text-books and handbooks that I have consulted. The nearest approach to a discussion of the subject that I have seen is in a valuable paper by C. S. Bull, in which the conclusions of Fournier as to the prophylaxis of hereditary syphilis in general are quoted. And yet the subject is one which should receive careful consideration, by the oculist and syphilologist as well as by the general physician.

By interstitial keratitis, in this paper, I refer only to the typical form, which occurs almost always between the ages of three and sixteen years, and almost never after the twenty-fifth year. The more I see of this disease the more convinced I become that Hutchinson is right in attributing practically all of it to inherited syphilis. I know that Panas and Fournier place the ratio at only about 41 per cent. I know also that this disease is caused by tuberculosis more often than was formerly supposed; also that Sedan[2] asserts that in Algiers it is more often caused by malaria than by syphilis; also that Neal[3] has frequently seen it in

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented at the Atlantic City Session, June 8-11, 1909. Publication rights reserved by the American Medical Association.

1. Med. Rev., April 4, 1908.
2. Abstr. in Arch. f. Augenh., 1888, 4, p. 443.
3. Ann. Ophth., April, 1908, p. 369.

China apparently as a result of severe diarrhea; but, on the other hand, Mauthner and Silex find the ratio of syphilitics to be 80 per cent. and 83 per cent. respectively; and, in a recent careful study of the subject, Stephenson[4] of London finds a ratio of 69.3 per cent. In the agricultural region in which I practice, where tuberculosis in children is relatively rare and the average nutrition rather high, I find other well-marked signs or histories of inherited syphilis in approximately 95 per cent. of my cases of interstitial keratitis;[5] and when we consider how often it is impossible to get a history in direct infection, I believe it is safe, in the absence of other obvious causes, to make a tentative diagnosis of syphilis in all cases of interstitial keratitis in patients between the ages of two and twenty-five. This, of course, does not mean that syphilis is unusually common in this region, but simply that the other causes of impaired nutrition are relatively inactive. For myself, then, the problem of the prophylaxis of interstitial keratitis resolves itself, for practical purposes, into the prevention and cure of syphilis, particularly of the hereditary form—a field so wide that only a few of its features can be treated here. With the prevention of syphilis in general, with the exclusion of syphilitics from matrimony, with the treatment of the parents, this paper need not concern itself. These points are amply considered in general works. Nor can I attempt to discuss any large part of the diagnosis of hereditary syphilis. I shall merely lay stress on certain peculiarities of the teeth as aids in diagnosis, on the treatment of the disease when it has broken out in one eye and on the prophylactic management of children marked with the disease but without any active manifestations of it.

THE TEETH AS AIDS IN DIAGNOSIS

To take up the last-mentioned phase of the question: I think it is highly probable that if all children between the ages of three and sixteen could be examined for the more obvious signs of inherited syphilis, and if those showing such signs could receive a long-continued course of specific treatment, controlled if possible by the serum diagnosis test, the number of cases of interstitial keratitis and of syphilitic deafness could be greatly reduced. Such examinations should be conducted by the physician in all families in which any member of the family has a history or symptoms which arouse a suspicion of syphilis. In public institutions all children should, on entrance, be inspected with this point in view, and when positive signs are found the appropriate treatment should be carried out. even if the child is otherwise apparently healthy. This recommendation as to treatment may need qualification if it turns out that the serum diagnosis is reliable in inherited syphilis. If this proves to be the case the treatment may perhaps be safely omitted in those showing repeatedly a negative result of the serum test; but even then the latter would have to be repeated at intervals of several years.

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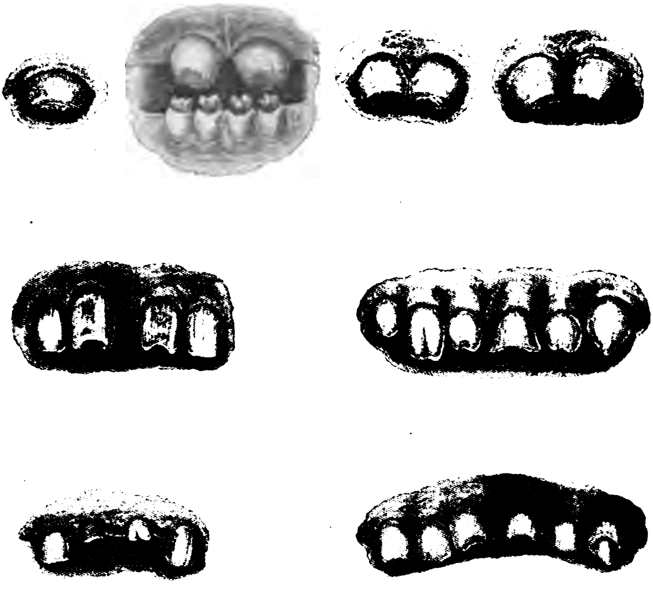
[4] Reprint from Med. Press and Circular, Dec. 25, 1907.

[5] In this estimate I exclude traumatic insterstitial keratitis (known in one of its forms as disciform keratitis), as well as certain one-sided forms of the disease which occur in adult life.



Figs. 1-8. "Hutchinson" teeth; lobed and notched incisors.

In the diagnosis of inherited syphilis the ordinary signs are well enough known. An excellent description of these and of many less common signs is given by

Fournier.[6] I merely wish, in this connection, to emphasize the importance of certain dental peculiarities. The old well-worn Hutchinson incisor is often scandalously misrepresented in otherwise excellent text-books. To give a fair idea of the Hutchinson teeth no one picture can suffice. To illustrate this I reproduce some of the figures (Fig. 1-10) from Hutchinson's original papers.[7]

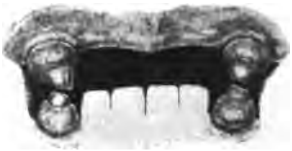


Fig. 9.—Small, discolored incisors with healthy canines.
Fig. 10.—Milk canines in which a discolored blunt peg projects from a base of normal-looking tooth tissue.

These central permanent upper incisors, on their first appearance, show instead of a notch, a central irregular lobe, as in Fig. 2, which generally wears away soon but which sometimes persists for several years. Later on the notch itself is obliterated by the rapid wearing away to which such teeth are subject. Hutchinson says that the notch is never seen after the age of

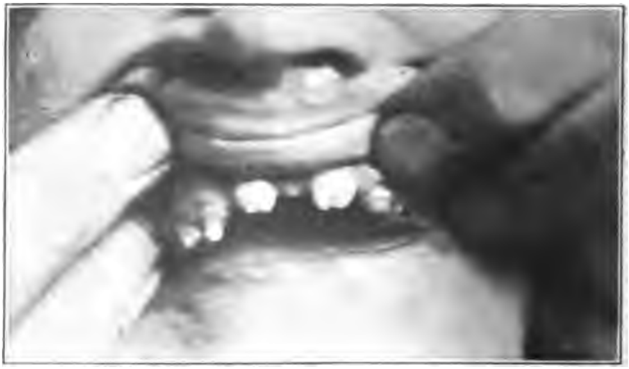


Fig. 11.—Girl, aged 8 years, subject of interstitial keratitis. Note characteristic permanent incisors and equally characteristic temporary canines. This girl also has well-marked Fournier molars.

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[6] Fournier, Edmond: L'hérédo-syphilis tardive, Paris, Masson & Cie.

[7] Tr. London Path. Soc., ix and x.

twenty-five or thirty, but I can not recall a case in which it could be seen well after the age of fifteen. When the notch is gone the character of the tooth can still be recognized in many cases by the fact that the lateral lines after bulging slightly, half-way between the gum and the edge of the tooth, converge slightly till they meet the cutting edge. This bulging of the lateral lines I consider the most permanent feature of the Hutchinson tooth. With regard to the milk teeth, Hutchinson, in his second paper, made some important observations which have practically passed into oblivion; at least I have not seen them mentioned by anyone else. One of these refers to the condition shown in Figure 9.

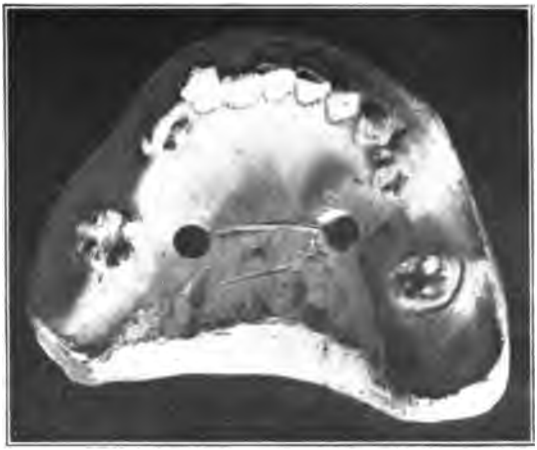

Fig. 12.—From 6-year-old child with interstitial keratitis. First permanent molar at the right is of the Fournier or Darier type.

in which the incisors are small and discolored while the canines are healthy-looking. This is a frequent condition in syphilitic children of the age subject to this condition. Figures 10 and 11 show another peculiarity of the milk canines on which Hutchinson lays great stress, and which I have seen a number of times. A central discolored blunt peg projects from and is separated by a shallow groove from a base or collar of normal-looking tooth tissue. If we imagine the same sort of defect occurring in a molar tooth we shall have a defect on which great stress has been laid by Darier, although it was, according to Suker, first described by the elder Fournier. Darier[8] describes these syphilitic first permanent molars as having the summit formed by four tuberosities or tubercles of a yellowish color surrounded by a groove separating them from the whiter enamel-covered body of the tooth, which bulges out on all sides beyond the tuberculated crown. These tubercles soon wear down or become carious so that unless seen in early life one finds simply a carious crown (or ,as Edmond Fournier[9] says, if caries does not set in, such teeth later on present a characteristic flat summit devoid of the normal cusps). Figure 12 shows the cast of such a molar from one of my own patients. Figure 13 is a reproduction of the cut accompanying Darier's paper. It shows, instead of four tubercles, which he describes, a central irregularly crenated mass, a form also described by Fournier.

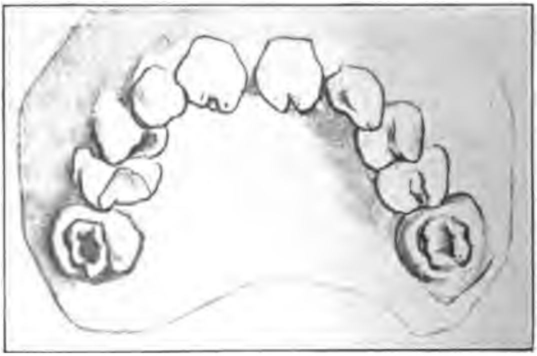

Fig. 13.—Reproduction from Darier's paper showing characteristic permanent molars with central discolored mass crenated, instead of tuberculated.

I consider these Fournier molars and the peg-shaped milk canine of Hutchinson to be fully as characteristic of syphilis as the Hutchinson incisor, and their recognition is of the greatest importance, since they often can be seen either together or separately at an age when the permanent incisors are not to be seen. The only mention of the tuberculated first molar that I have seen outside the writings of Fournier and Darier is in Suker's paper.[10] Another form of syphilitic tooth on which Darier lays stress is the tuberculated permanent canine shown at the right of Figure 8 and in Figure 14. Hutchinson also mentioned this form but did not consider it so characteristic of syphilis as did Darier. I

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[8] Bull. et mém. Soc. franç. d'opht., 1903, p. 216.
[9] L'hérédo-syphilis tardive, p. 100.
[10] Reprint from Medicine, September, 1904.

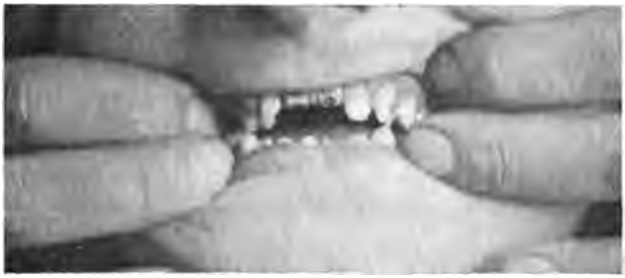

Fig. 14.—Girl of 16 years with interstitial keratitis. Hutchinson central incisors unusually small. Note also, at the right, permanent tuberculated canine.

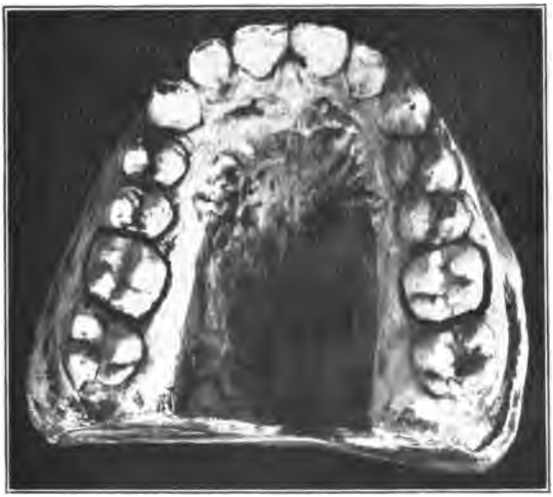

Fig. 15.—"Sloped" permanent molars in boy of 14 years, of robust build and good previous health until puberty, when he developed characteristic bursitis of right knee joint, interstitial keratitis, and necrosis of hard palate. Younger sister, a year later, had similar train of symptoms; teeth otherwise normal.

have only seen a few examples of it, but they have all been in patients with other well-marked signs of syphilis.

There is one other form of first permanent molar which I have observed in children with interstitial keratitis and other signs of congenital syphilis, and which I have not seen mentioned by any author. This is what I call the sloped molar, shown in Figure 15. In this form the base of the tooth as it emerges from the gums is much wider than the crown, the cusps being fairly well formed, with little or no tendency to decay. I have seen it only half a dozen times but in all these cases it was of some importance, as the other teeth showed nothing decidedly wrong. It is probably due to the same influences of malnutrition which when acting more decidedly produce the Fournier molar.

TREATMENT

As to the prevention of the disease in the second eye, after it has once appeared in one eye, the sentiment appears to be universal that treatment has but little influence in this respect; and for many years, while under the influence of the idea that interstitial keratitis was, at most, a parasyphilitic manifestation of malnutrition, I used the specific treatment with moderation only, and invariably obtained negative results in the attempt to prevent the affection of the second eye; in fact, the immediate effects of antisyphilitic remedies were so slight that I placed little reliance on their use; but since the discovery of the spirochete, the evidence that the disease is due to an active invasion of this germ has become so overwhelming that the necessity for radical specific treatment is patent.

For the last three years I have treated interstitial keratitis with the same vigorous course of inunctions which I should use in an ordinary syphilitic iritis, having a piece of the mercurial ointment about the size of the patient's thumb (that is, for a man of 150 pounds weight, about 275 grains of the ointment) rubbed in for at least half an hour every day for a week, unless the gums or teeth get sore before that time is up. This course is repeated after a week's rest and then for one week out of every three for the next two to four months, according to the severity of the case; after that the period between the courses is lengthened to from four to six weeks. This is kept up for a year, and for the second year the course is taken for one week out of every two months. All through the active course of the disease atropin and hot applications are used vigorously, and enough salicylate of sodium is given to control the severity of the symptoms of congestion and photophobia. For a full-grown patient from 90 to 150 grains a day for a few days is generally sufficient to break the force of the inflammation, and after this, 90 grains a day on three or four days in the week is generally sufficient. After the congestion has been entirely absent for three weeks, the atropin and salicylate are discontinued and not used again unless the congestion reoccurs. In the interims between the courses of mercury I have used arsenic and iodids in moderation. I have a decided impression that I have been obtaining better results since adding the arsenic to the treatment of this disease, but whether it is best to give it in the form of injections of atoxyl, or some other of the newer preparations, or by the mouth in Fowler's solution, I am not prepared to say Since adopting this line of treatment I am certain that my general results have been decidedly better than they formerly were, and I have seen three typical cases in which the disease has failed to appear in the second eye during periods varying from one to three years. These may be mere coincidences, and I know what long periods are sometimes reported as occurring between the infections of the two eyes, but the fact that in twenty-three years of eye practice I have never, before, seen an attack in the second eye postponed longer than three or four months, gives me a decidedly hopeful feeling as to the possibility of preventing the disease in the second eye when the first eye is seen early in the attack.

SUMMARY

To sum up and repeat, I believe that the following points may be made with reference to the prophylaxis of interstitial keratitis. First, in the text-books on ophthalmology and syphilis the diagnosis of hereditary syphilis should receive much more attention than it usually does; and, instead of the single faulty cut so commonly used, at least half a dozen figures should be presented, to show not only the Hutchinson incisors, but the other more important forms of syphilitic teeth. Second, all children in public institutions and in private families who show any of the well-marked signs of inherited syphilis should receive a course of antisyphilitic treatment, even if in other respects they seem to be entirely well; the results and indications of this treatment being controlled, if possible, by the serum diagnosis test. Third, where a case of syphilis, inherited or otherwise, appears in a family, all the other members of the family should be examined for signs of the disease, and if such are found, the bearer should be subjected to specific treatment. Fouth, by an extra-vigorous use of specific treatment the disease may be kept out of the second eye in a larger proportion of cases than has hitherto been thought possible.

867 Brandeis Building.

MEMORANDA

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617+

U. HOLZER
BINDER
BOSTON, MASS.